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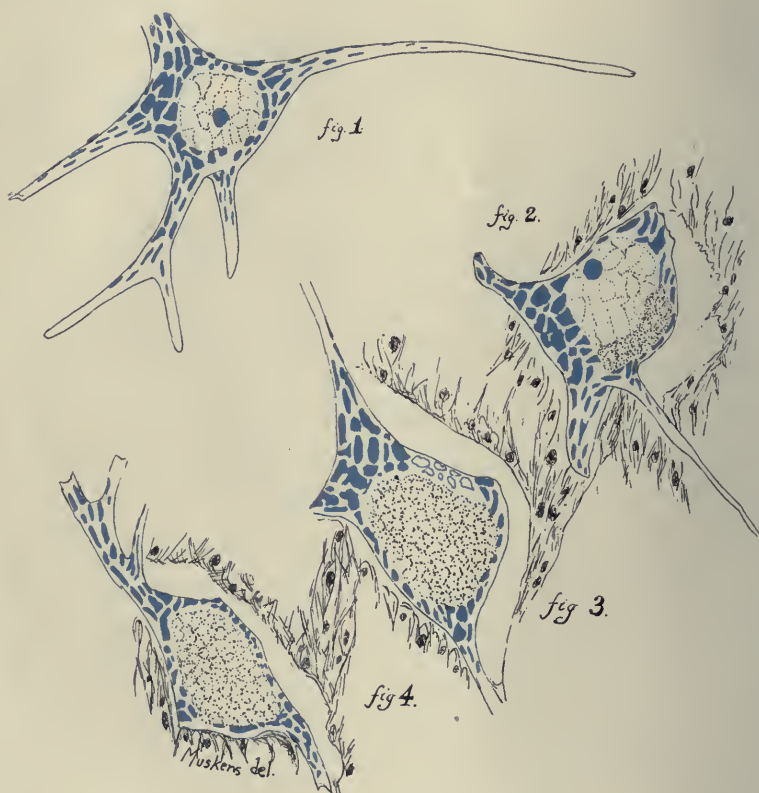


FIG. VIII. Fig. 1. Normal cell. Figs. 2, 3, 4, show cells of anterior horn in different stages of degeneration.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

SUB-ACUTE COMBINED SCLEROSIS OF THE SPINAL
CORD,¹

AND ITS RELATION TO ANEMIA AND TOXEMIA.

BY CHARLES L. DANA, M.D.

There is a group of cases that may be characterized broadly as forms of ataxic paralysis of subacute course. They are not so very uncommon, and it seems to me they should be better known as representing a distinct clinical type. The trouble is essentially a spinal one. It occurs chiefly during or just after middle life, and is associated with a cachexia, sometimes with pernicious anemia, and with other toxemic states. Attention was called to this condition first by Dr. J. J. Putnam, and then by myself seven years ago (*Journal Nerv. and Mental Dis.*, Feb., 1891, April 1891). A little later Dr. Grainger Stewart (*British Med. Jour.*, 1891,) reported a case evidently belonging to the group, quoting our papers. At least ten articles recording similar cases have been published since then, not including those specially belonging to pernicious anemia. The important papers were those of Leyden², Rothmann³, Teichmüller⁴, Boe-

¹ Read before the New York Neurological Society, Nov. 1st, 1898.
For discussion on this paper see page 37.

² Zeitschrift für klinische Medicin, Bd. 21, H. 1-2.

³ Deutsche Zeitschrift für Nervenheilkunde, 1895, VII.

⁴ Deutsche Zeitschrift für Nervenheilkunde, 1895, VIII

diker and Juliusburger⁵, Bowman⁶, Nonne⁷, Rhein⁸, Mitchell and Rhein⁹. The whole subject was carefully monographed in 1895 by Rothmann and in 1896 by Bastianelli¹⁰. None of these writers, however, except the last named seem to have seen the articles by Putnam and myself. Most of them, in studying this subject, have been intent on the anatomical findings, and have tried chiefly to establish a specific organic change in the columns of the spinal cord. Their collections of cases have been confusing on account of the variety of the clinical pictures associated with the combined sclerosis.

It is my object especially to call attention to the fact that there is a definite disorder recognizable by its symptoms and course, as well as by the anatomical changes found after death. Dr. Putnam quite recently told me that he had seen a good many cases of the type since his original article. I have seen six cases in the last three years, and the disease is apparently more common than multiple sclerosis, and nearly as common as the spinal atrophies.

Much direct and side light has been thrown upon the nature and symptoms of the malady by a number of investigators who have studied the pathological changes of the spinal cord in pernicious anemia.¹¹

They found that in pernicious anemia there was sometimes, but not always, a pathological change in the spinal cord.

⁵ Neur. Centralblatt, 1896, p. 326.

⁶ Brain, Summer, 1894.

⁷ Arch. f. Psych., XXV., 1893. Deut. Zeitschrift f. Nervenheilkunde, VI.

⁸ Rhein, Jour. Nerv. and Ment. Dis., Nov., 1896.

⁹ Mitchell and Rhein, Jour. Amer. Med. Assoc., April, 1898.

¹⁰ Bull. d. R. Acad. Med. di Roma, 1895-6.

¹¹ In 1887, Lichtheim, Verhandlungen d. VI. Congress für innere Medicin, Wiesbaden, 1887, reported a series of cases of pernicious anemia, in which he described changes in the spinal cord. His investigations were followed by those of Eisenlohr, Deutsche med. Wochenschrift, 1892; Von Noorden, "Untersuchungen über schwere Anämien," Charité-Annalen, 1891-'92; Minnich, Zeitschrift für klinische Medicin, vol. XXI, Ma. 1-2, vol. 22; Nonne, Archiv für Psychiatrie, vol. XXV; Burr, University Medical Magazine, 1895; Lloyd, Journal of Nervous and Mental Disease, 1896; James Taylor, Medico-Chirurgical Transactions, vol. LXXVIII; and Riggs, International Medical Magazine, September, 1896.

This consisted of a degeneration which always affected the posterior columns, and in most cases both the posterior and lateral columns, but never the lateral columns alone. The gray matter and the nerve roots were rarely touched, and the change was of the nature of a degeneration of the nerve tracts without any later shrinking of the cord, as in locomotor ataxia. The blood vessels sometimes showed hyaline changes, but were not always much diseased, though small hemorrhages were now and then seen. These changes of the spinal cord peculiar to pernicious anemia were, as will be seen later, not strictly like those seen in the class of cases to which I now refer.

With this brief summary of previous work I proceed to relate the history of my own case:

Summary.—M., age 50. No syphilis, no lead, no alcoholism. Spring, 1897, numbness in right hand, then in left, ataxia and weakness. Later, girdle sensation, weakness in limbs, with numbness, emaciation. Progressive weakness, paraplegia, some anesthesia of lower and upper extremities, pains in back and legs, loss of control of sphincters, no severe anemia, death in one and a half years.

Autopsy.—Spinal cord, degeneration throughout whole length of posterior and lateral columns, confined mostly to columns of Goll and crossed pyramidal tracts. Recent acute softening in anterior horns and columns at lower cervical and first dorsal segments, same in lower dorsal segments. Anterior horns affected at some levels. Some disease of blood vessels, small hemorrhages.

History.—Converse L., native of Maine, 50 years of age, single; by occupation, an engineer. His father died of stricture of the esophagus, one brother died of some mental trouble following typhoid fever, and a sister had consumption. There is no history of any definite nervous disorder in the family. The patient had always been a healthy man, of good habits. He drank moderately. There was no history of syphilis.

In 1861 he had typhoid fever, and in 1875 he suffered from an attack of round worms. In 1886 a heavy pipe fell on his head and stunned him for about ten minutes. In other respects his previous history was negative.

About one year before he was seen by myself, and without any known cause he noticed that the fingers of the left hand became numb. This numbness soon extended to the right hand, but to a less degree. The numbness was, in the course of a few weeks, associated with some awkwardness in the use of the hands, and also with some actual weakness in the arms.

He was able to work, however, until about two months before admission. He then felt a sensation of a band around the waist, and developed a weakness in the lower extremities, which was also accompanied with some numbness and with some wasting of the muscles. This anesthesia and weakness of the arms and legs continued until he became unable to walk, and he was admitted to Bellevue Hospital on Jan. 3d, 1898. At that time he could stand, but not walk; he could use his hands with difficulty on account of the ataxia rather than weakness. He complained of pain in the arms, neck and back, and of sensations of coldness and of muscular twitchings in the legs. He suffered from insomnia. An examination of the lungs, heart, abdominal viscera and urine showed no morbid changes. The urine was acid, clear, its sp. gr. was 1.028; it contained a trace of albumin, but no sugar. There was a general emaciation, and he said that he had lost about thirty pounds in the last few months. He was anemic, but not strikingly so.

Examination.—There was a paresis of the lower extremities, especially of the right. The extensors and flexors of the right leg were almost entirely useless, and those of the left were very weak. The muscles of the right thigh were almost entirely paralyzed, so that he could with difficulty draw up or extend, abduct or adduct, the thigh. This paralysis involved more especially the right thigh. There was weakness of the arms, but he could move them in all directions, the right arm being much the worse. His co-ordination, however, was very bad. The grasp of the dynamometer in the right hand was 45, in the left 60. The circumference of the forearms was only 6 inches, and that of the calves only 10 $\frac{3}{4}$ inches. The kneejerks were present and exaggerated. The superficial reflexes were also exaggerated. There was a diminution of the galvanic reaction of the muscles of the legs and arms, but no degenerative reaction.

In the feet and legs tactile sensation was blunted and delayed, muscular sense lost and temperature sense inaccurate. In the arms tactile sense was delayed and blunted, but not to a great extent. Temperature, pain and muscular sense, however, were very much disordered, and the loss of muscular sense, with consequent ataxia, was most marked of all. His pupils reacted to light and accommodation, no nystagmus was present, visual acuity and fields of vision were normal, hearing was slightly impaired, taste was blunted, smell was normal. He had no disturbance of speech or swallowing. His temperature was normal; his pulse ranged from 72 to 90, respiration from 18 to 20.

The general symptoms, therefore, were those of a patient

suffering from a somewhat spastic and ataxic paralysis of the legs, with marked ataxia and some weakness of the upper extremities.

The anesthesia involved the upper extremities, reaching to near the shoulders, and the legs, reaching to the lumbar region, but not the trunk. The tactile anesthesia was slight, the temperature and pain anesthesia greater, the muscular anesthesia most marked of all, especially in the arms. The earliest and most marked characteristic sensory disturbance was the numbness and ataxia in the arms. This was followed by tactile anesthesia, and then by a moderate amount of muscular weakness in the arms.

The next set of symptoms were those involving the legs and feet; the sensory symptoms appeared first, followed quite rapidly by weakness of the legs with stiffness, exaggeration and then lessening of the reflexes, and finally by a nearly total paralysis.

The patient looked emaciated and very slightly anemic. He had, however, no striking blanching of the skin or mucous membranes, not enough, at least, to attract attention or lead to an examination of the blood. He had no hemorrhages at any time, and no special disturbances of the stomach or intestines. His mind was clear until the last week of his illness, and he showed no signs then of any loss of memory or dementia. He had no convulsive seizures of any kind. His paralysis was not accompanied with any tremors or any fibrillary twitchings. He had considerable pain in the back and limbs.

The patient continued under observation until Feb. 8, a period of over six weeks. During this time the paralysis of the legs became greater until it was nearly complete. The anesthesia increased slightly. He lost control over the bladder. The condition of the arms did not change so much, but there was a steady decrease of power. He suffered during this time from pains in the legs and back, but the mind continued clear, and there was no involvement of the special senses, other than that mentioned.

He was given iodide of potassium, arsenic and iron, and was treated symptomatically for his pains and insomnia with codeine and trional. He gradually became weaker; on Feb. 7th his temperature began to rise, and he became entirely unconscious. On Feb. 8th the temperature rose to 102 deg., pulse to 108, and he died from exhaustion.

Autopsy.—The autopsy was made by Dr. C. Phillips, of the Carnegie Laboratory.

The specimens of the brain and cord were placed in alco-

hol and Müller's fluid, and sections were made and stained with hematoxylin, carmine and by Nissl's and Weigert's methods.

The results of the autopsical examination outside of the nervous system showed nothing of great importance, the changes being mostly such as would be found after great emaciation. The muscular system was fully developed, the skin pale and unelastic and with but little subcutaneous fat. The lungs showed no tuberculosis, but were adherent to the chest wall. The heart was small, the left ventricle contracted, weight 9 oz. Some atheroma of the coronary arteries was found. In connection with the subject of pernicious anemia, it is important to note that the stomach was very small, having a capacity of only about 300 cubic centimeters, and measuring only 16 centimeters in length. The mucous membrane was normal. The small intestines and mesenteric glands were normal. The spleen was enlarged and the kidneys also were enlarged, but showed no other change than a congestion. The suprarenal bodies were normal. The liver weighed 2,095 grams, but showed nothing abnormal, though Quincke's test was not tried.

The skull cap was symmetrical and showed rather deep but regular markings. The brain membranes were normal. The brain itself was of average size and the convolutions symmetrical and normal in appearance. There was some increase in the intraventricular fluid. The blood vessels at the base of the brain showed a few areas of arterial sclerosis. These were limited to the circle of Willis. The brain otherwise appeared normal.

The Spinal Cord.—On opening the spinal column the dura mater appeared quite pink in color, slightly thickened and partially adherent over the cervical and lumbar portions. Its blood vessels were normal. The pia mater appeared to be markedly congested in the area reaching from the fifth to the eighth cervical and from the fifth to seventh dorsal regions; also in the lower lumbar. In the sacral regions were a few small areas of subpial hemorrhage. The substance of the cord seemed firm, except in the region of the eighth dorsal segment, where it tended to assume a flattened appearance. In the first, third and fifth sacral segments were small grayish-white masses which were differentiated from the balance of the cord. They varied in size from one to two centimeters in diameter and were firm in consistency.

Microscopical examination showed a brown atrophy of the heart muscle, a chronic diffuse nephritis, and a simple atrophy of the liver with pigmentation.

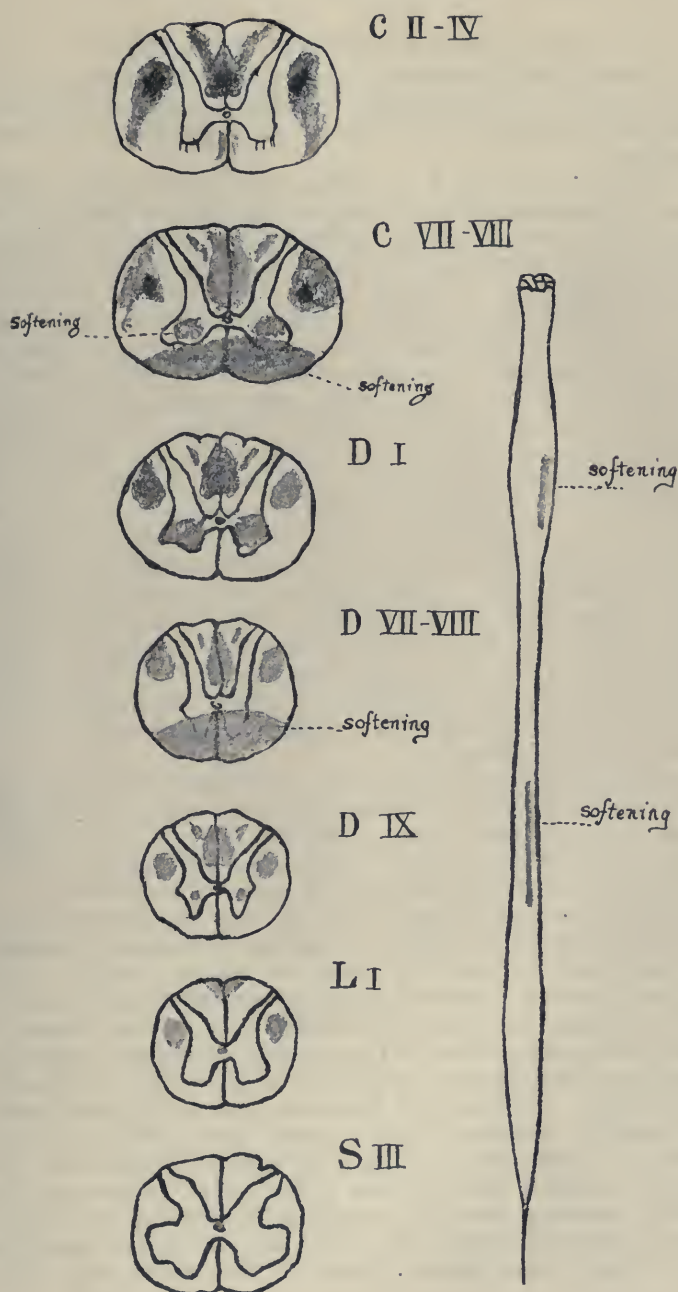


FIG. 1. Showing the foci of softening and the location of the systemic degeneration.

MICROSCOPICAL EXAMINATION OF CORD.

Sections of the cord were made at the level of the 2, 4, 6, 7, 8, cervical; 1, 4, 7, 8, 9, dorsal; 1 lumbar and 3 sacral segments. They were stained by Dr. Phillips, to whose help I am much indebted.

Second Cervical Level.—Pia mater practically normal. Anterior columns; (Fig. II) slight degenerative change in the left anterior median column. Lateral columns, marked vacuolar degeneration in the region of the pyramidal tracts, extending to the periphery. Very few nerve fibers can be seen here; only a wide-meshed reticulum of connective tissue; a degeneration of the antero-lateral ascending tracts. Direct cerebellar tracts not affected. The lateral limiting layer shows occasionally a few vacuolar spaces. Posterior columns show a degeneration involving the deeper parts, including the columns of Goll, but leaving comparatively untouched the V-shaped area near the periphery. The root zones are not affected. The gray matter of the anterior horns is moderately healthy, but some change can be seen in the nerve cells (Fig. II). The anterior and posterior roots show some loss of nerve fibers, but no extensive change. The blood vessels of both the gray and white matter show thickened walls, with what I take to be a *hyaline change* in them. There is, undoubtedly, an increased vascularity of the whole section of the cord; in some places the blood vessels are dilated and in others collapsed, and here and there are a few minute extravasations, but there is *no evidence of true inflammation*.

Fourth Cervical.—(Fig. III) The changes here resemble those just described, but are much more marked. The vascular change appears more active, and there is more involvement of the anterior horns. The oldest process at this level seems to be in the deep parts of the posterior columns.

Sixth Cervical.—(Fig. IV) No decided change can be made out in the anterior median or anterior fundamental columns. In the lateral columns the change is confined chiefly to the pyramidal tracts, spreading thence apparently to the periphery and involving the direct cerebellar tracts. The process is most serious and oldest in the posterior columns which have undergone throughout nearly their whole extent a degenerative change. The anterior horns are also at this level partly destroyed by softening, leaving empty areas. Where the gray matter is left one can still find good anterior horn cells. The vascular changes here are also rather active. One sees long vessels reaching from the meninges directly to the anterior horns, where they apparently end in dilatations, and sometimes in punctate hemorrhages. Some of the vessels have thick

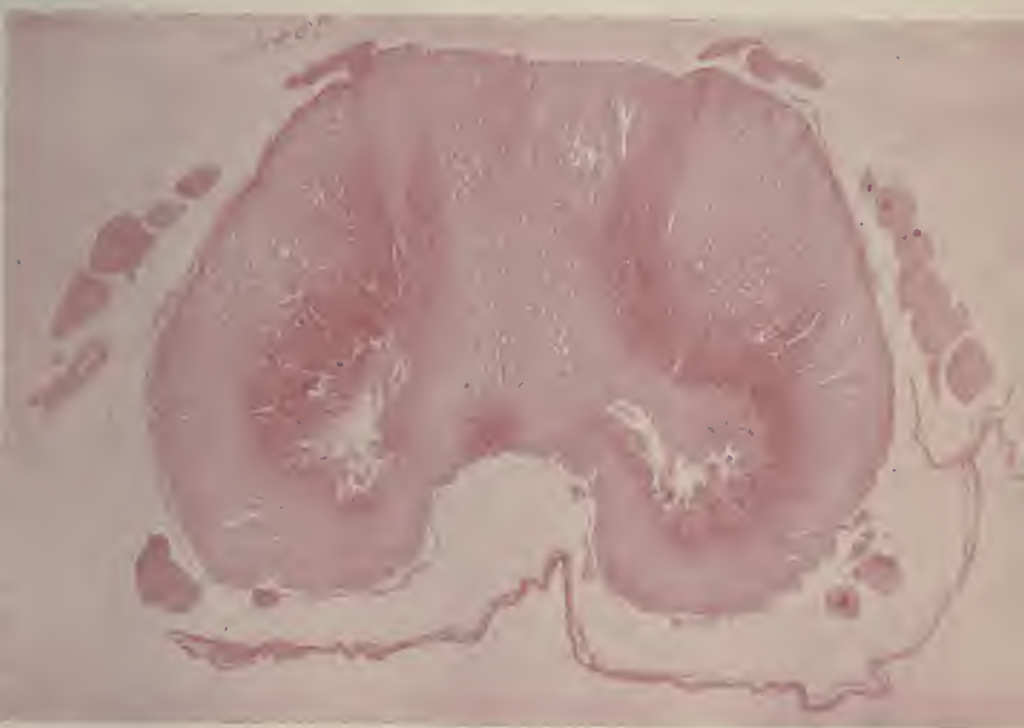


FIG. IV. Sixth cervical segment. The anterior horns are partly softened, leaving a cavity.

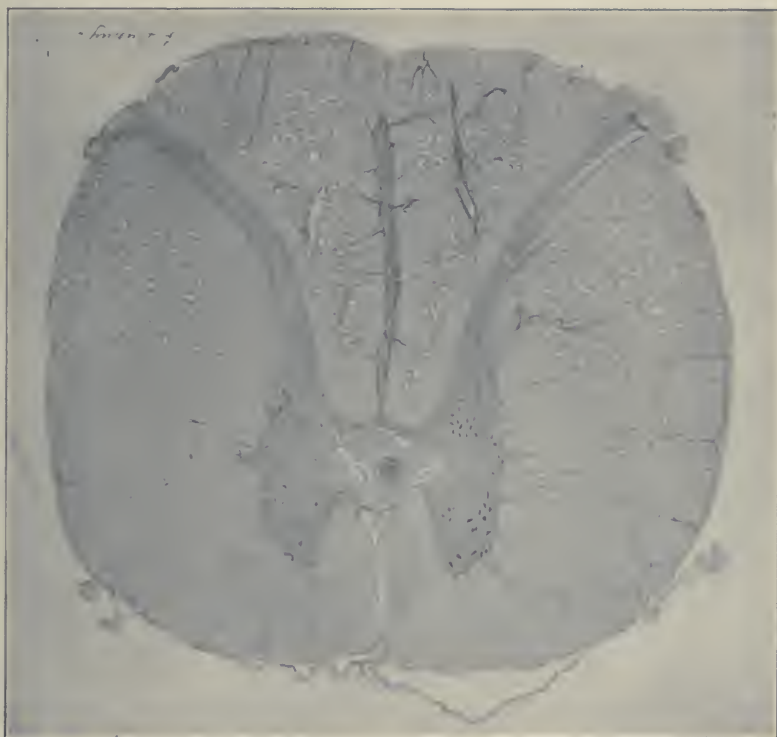
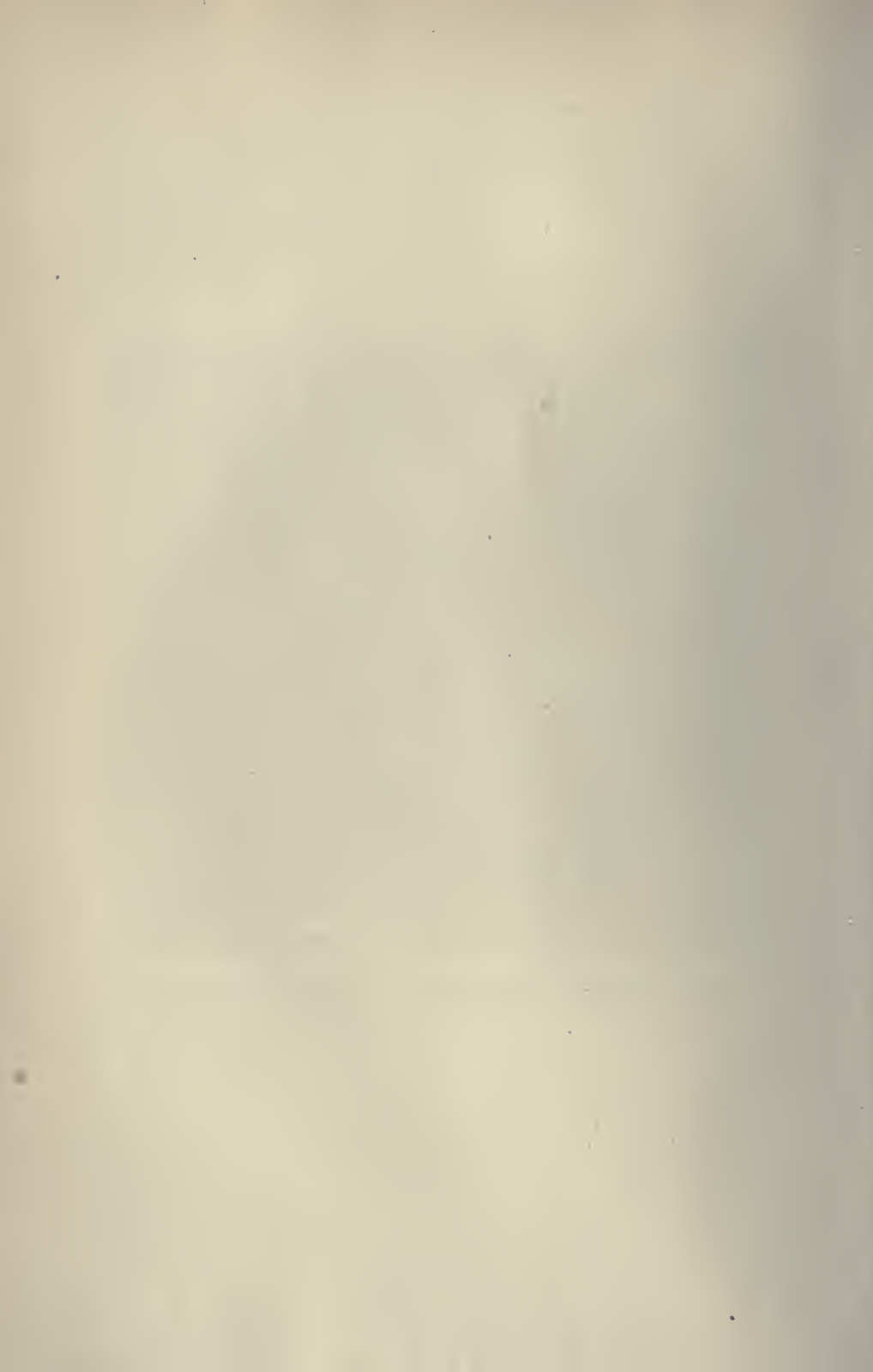


FIG. II. Second cervical segment. The degenerated areas are indicated by the vacuolated appearance of the section.



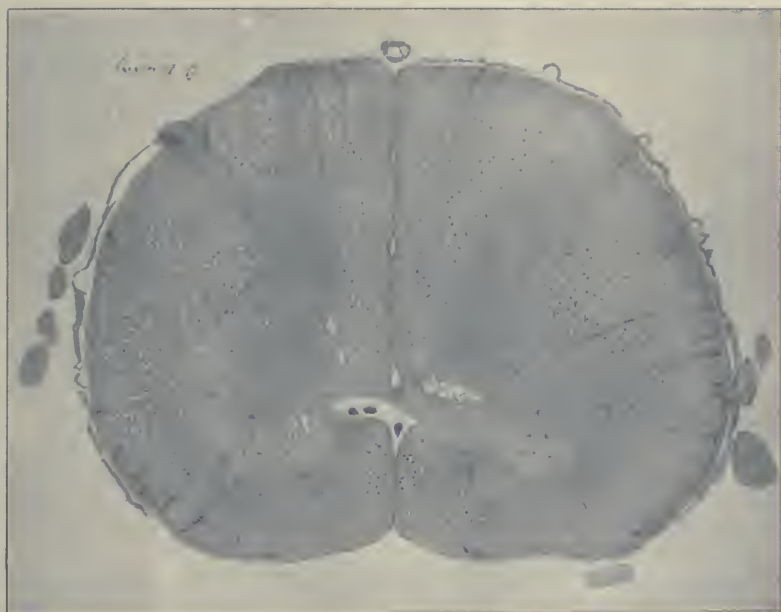


FIG. III. Fourth cervical segment. (The reproduction does not show very perfectly the intensity of the process.)

walls and look collapsed. The picture suggests the possibility, therefore, of a *softening* in part at least, from *thrombosis*.

Seventh and Eighth Cervical.—Here a large part of the anterior columns and part of the lateral columns have been destroyed or broken away. The loss of these parts, as shown in the figure (Fig. V), is probably due, however, to a difficulty in manipulating the softened cord at this point, and not due to the fact that there was an actual primary softening in this region. Weigert's stain at this level shows that the degeneration, though very marked and striking, is limited in the lateral columns almost entirely to the crossed pyramidal tracts, extending slightly into the lateral limiting layer. The direct cerebellar tracts and Gowers' tracts are not affected. The pos-

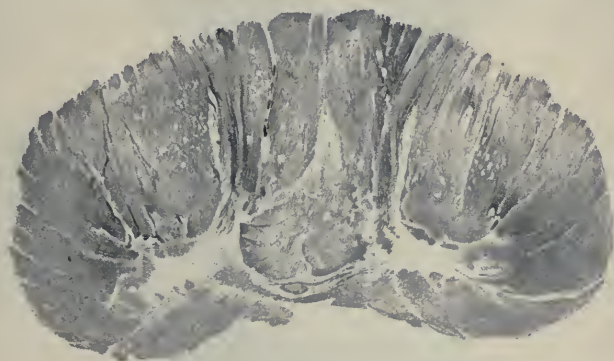


FIG. V. Eighth cervical segment.

terior columns are very extensively involved, but areas of normal fibers are found here in the posterior fundamental column and in the root zone on each side. The blood vessels here also show thickened walls and appearances similar to the sections higher up. The *anterior roots* show degeneration of fibers.

First Dorsal.—Here the change is similar to that in the lower cervical, but less marked. The anterior horns are so badly softened that they have dropped out, leaving open spaces. The degeneration in the lateral columns is still mainly confined to the crossed pyramidal tracts. Here the sclerosis is dense in its central part, and one sees numerous dilated and tortuous blood vessels, but no hemorrhages, and no proliferations or exudations. The direct cerebellar tracts and Gowers' columns are not affected. The area of greatest activity of the pathological process is still deep in the posterior columns.

Fourth Dorsal.—This shows again a sclerosis affecting the lateral and posterior columns as much as in the first dorsal, but with less intensity. The anterior horns are also less in-

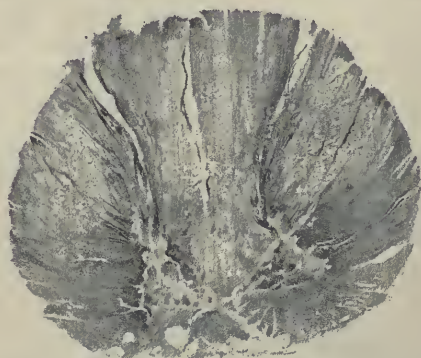


FIG. VI. Fourth dorsal segment.

volved. The cells of the columns of Clarke are somewhat affected, but apparently only secondarily. In the anterior horns there is a great deal of active vascular change and several *small hemorrhages* (Fig. VI).

Sixth to Seventh Dorsal.—The anterior horns here, in fact, the anterior third of the cord, have fallen in as a result of

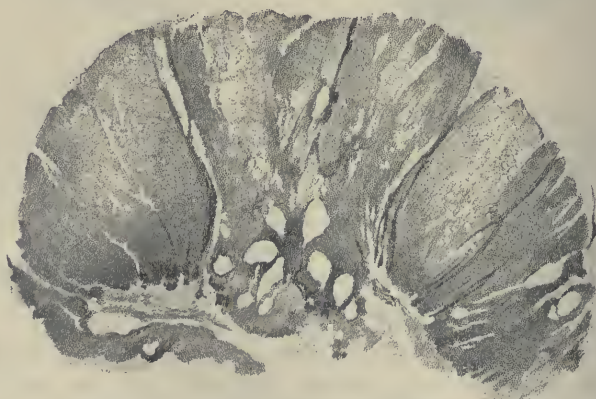


FIG. VII. Sixth to seventh dorsal segment.

softening and post-mortem manipulation. (Fig. VII) The degenerative process in the remainder of the column shows more activity than was present in the segment of the dorsal cord

above. The change is more extensive in the lateral columns, but even here it does not reach the periphery, and does not involve the cerebellar or Gowers' tracts. But the softening is more profound and the vacuolar spaces more extensive. Similar evidence of the more active and extensive degeneration is present in the posterior columns. The cells of Clarke's columns are normal on one side, but have been destroyed on the other. Widely dilated vessels and small hemorrhages can be seen in the anterior horns, parts of which have softened and dropped out. The anterior horn cells, however, which remain are in fair condition. The anterior roots show in their centers large dilated vessels, but no very extensive degeneration. Neither here nor above has there yet appeared *any sign of meningitis*. The vessels do not seem to have as much thickening of the walls as in the cervical region. Examples of the degenerating anterior horn cells (Nissl stain) are shown in Fig. VIII.

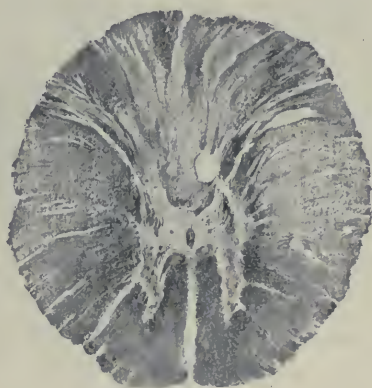


FIG. IX. Ninth dorsal segment.

Ninth Dorsal.—Here the degeneration is most marked in the posterior column, especially the posterior and median. It is present in the lateral columns, but moderate in intensity, reaching, however, to the periphery, and to some extent perhaps involving the direct cerebellar tracts, but not the tracts of Gowers. Lissauer's columns are normal. Small hemorrhages can sometimes be seen in the sclerotic parts here, and other sections show the presence of numerous amyloid bodies (Fig. IX).

First Lumbar.—Here there is a slight sclerosis confined to the crossed pyramidal tracts, and a very slight change also in the peripheral portion of the posterior columns in their median

part. The anterior horn cells here are normal. The third sacral segment is practically normal (Fig. X).

To summarize the foregoing we find that there were two original foci of disease, one in the lower cervical zone and one in the lower dorsal region, the upper being the older. The earliest part attacked was the posterior columns, especially in the central and deep parts. The process extended to the posterior external columns at some levels, but never entirely involved the root zones. The lateral columns were badly diseased also, especially at the two levels mentioned. The crossed pyramidal tracts were the most af-

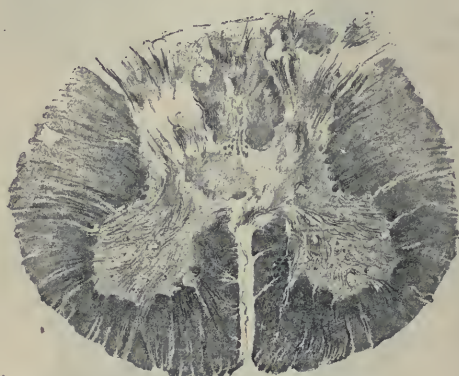


FIG. X. First lumbar segment.

ected, but the disease extended at the cervical level to the direct cerebellar tracts and Gowers' columns. Below, the disease was limited to the crossed pyramidal tracts so sharply that it might be called secondary, but the microscopical appearances were almost too active and acute for this. The change in the pyramidal tracts was much less noticeable high up in the cervical region, and could not have been secondary to any cerebral or bulbar process, though other parts were not examined microscopically. They were macroscopically normal. The gray matter of the anterior horns was seriously diseased, and in parts entirely softened. (Figs. XI and XII.)

Shortly before death there was a central softening, involving the anterior horns and part of the anterior columns at the level of the lower cervical and mid-dorsal segments. It was

just such a process as might be produced by a thrombosis of the anterior median artery. Microscopical examination showed this process to be a degenerative change, in which the nerve fibers were in great part destroyed, leaving large vacuoles (vacuolar degeneration). This was followed in the older areas by proliferation of connective tissue, and a true sclerotic change. In some levels there were small hemorrhages into the anterior

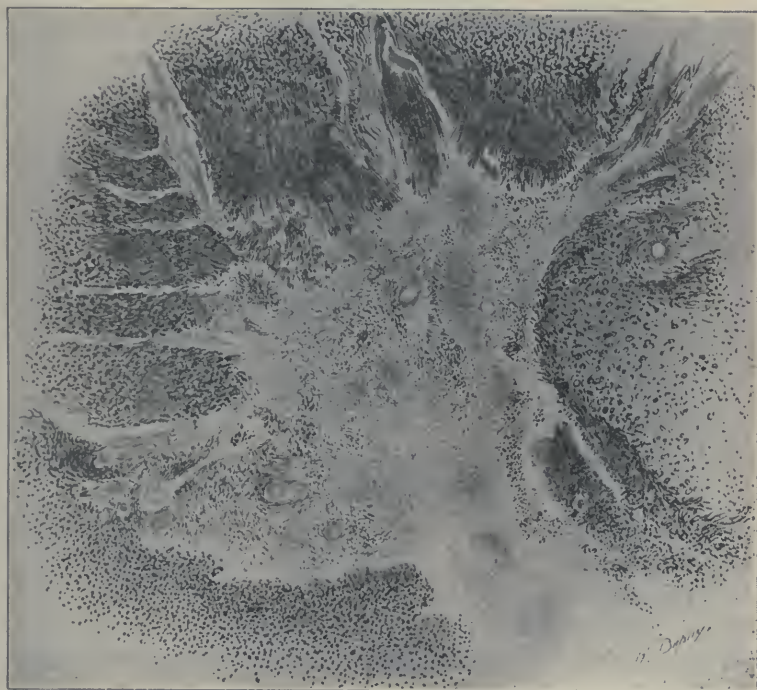


FIG. XI. Anterior horn of fourth dorsal segment, showing smogging. The diffuse smoky areas in the inner and lower part are capillary hemorrhages. ($\frac{2}{3}$ objective.)

horns. The blood vessels, in parts, were certainly diseased. This vascular disease involved the median and outer coats, especially the former, and seemed like a hyaline thickening. It was not like the peripheral periarteritis of syphilis. (Fig. XIII.)

REVIEW OF CASES AND HISTORY OF THE DISEASE.

Several collections of cases of combined sclerosis have been made in the last twelve years, and attempts naturally

followed to make out the description of diseases due to such scleroses, yet not belonging to paresis, tabes, hereditary ataxia, chronic myelitis or disseminated sclerosis¹². I tried this myself eleven years ago, but came finally to the conclusion that nothing very definite could be recognized, and that most, if not all of the cases called by Gowers "ataxic paraplegia," were

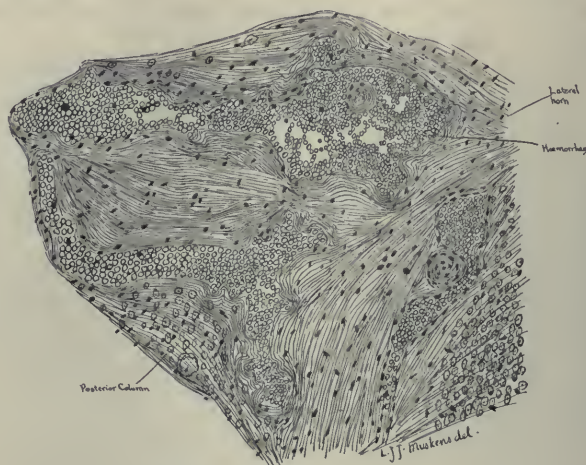


FIG. XII. Showing capillary hemorrhages in the anterior horn of the fourth dorsal segment. ($\frac{1}{10}$ objective.)

aberrant or complicated forms of tabes, transverse myelitis or multiple sclerosis.

The time has come, however, when this view need not be taken. There seems to be without doubt a disease which has features of its own, both clinical and anatomical, and which need not be confounded with those above mentioned. I have analyzed seventeen such cases reported with autopsies. This, perhaps, does not include all, but I have taken those which seemed to me to represent the type. I have excluded, for example, a case of Rothmann occurring in a syphilitic person and two of Boediker and Juliusberger occurring in the insane. Rothmann tabulates twenty-seven cases, but he includes, in my opinion, irregular, complicated or doubtful forms. I find that

¹² Omerod, *Brain*, April, 1880; Grasset, *Archives de Neur.*, 1886, Nos. 32, 34; Dana, *Medical Record*, July 2d, 1897; Rothmann, *Deut. Zeitsch. f. Nervenh.*, 1896.

but one sure case among Nonne's collection. Eight cases were reported by American neurologists (*loc. cit.*).

Etiology.—The disease occurs oftener in men, according to statistics, but my clinical experience is against this. Four out of my six cases were women. The age of most cases is between 50 and 60 (9 out of 17), and next to this between 40 and 50 (5). The youngest case was 36 (Rothmann's). There is often some family or personal neuropathic history.

The disease follows acute infections like influenza, prolonged diarrheal or dysenteric attacks, possibly shock, lead

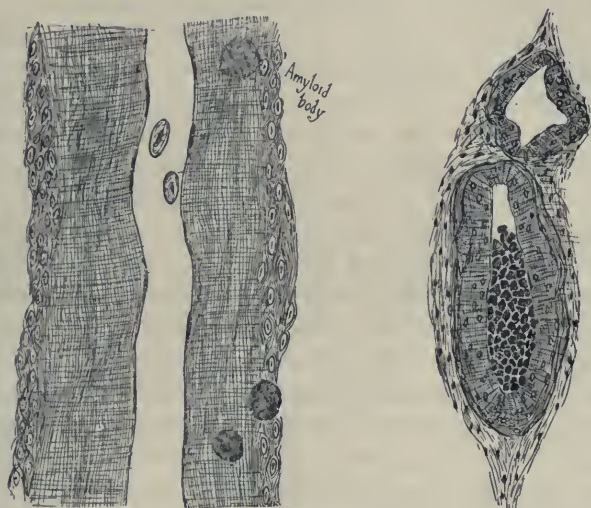


FIG. XIII. Showing cross-section and longitudinal section of small arteries. The specially thickened middle coat should be represented as quite homogeneous.

poisoning, malaria, lathyrism (?) and pellagra (?). One patient had a malignant tumor. Syphilis is not a cause. The relationship to profound secondary and to pernicious anemia is very important. In about 10 per cent. of cases, and perhaps more, pernicious anemia undoubtedly exists; in others there is usually a very marked anemic and cachectic state. Often, if not always, however, nervous symptoms develop before evidences of extreme or extended anemia are present.

Symptoms.—The initial nervous symptom is nearly al-

ways a persistent paresthesia usually of the feet, associated with some weakness of the part. Following this there is a good deal of ataxia, and this symptom increases with the loss of motor power. Tactile, thermic and pain anesthesia are not marked until late in the disease, but the patient often has severe pains in the back and limbs. There may be some differentiation of sensibility as in syringomyelia. With the weakness of the legs there is at first an increase of knee-jerks, and sometimes ankle clonus and rigidity, so that the patient shows the symptoms of spastic and ataxic paraplegia. Later the spasticity may become less and the knee-jerks disappear.

The arms are involved after a few months, the symptoms beginning with paresthesia, weakness, awkwardness and a very slight anesthesia. The disease may begin in the arms, as in my second case. The mind may be slightly weakened, the memory becoming defective and the patient emotional. The cranial nerves are not often involved, though optic atrophy has been once noted. Limitation of visual field occurred in one case seen by me with Dr. Markoe. The blood presents evidences of anemia. Usually this is secondary, but in a considerable number of cases the typical findings of pernicious anemia are present. The following records are taken from a typical case of combined sclerosis; the patient, a woman of about 60, having eventually pernicious anemia. The first examination was made in the early stage (first 6 months) of the disease; the last when the patient had become nearly paraplegic (1 year). The examinations were made by Dr. Frederic E. Soudern at the request of Dr. Frank H. Markoe, who had charge of the patient, and to whom I am indebted for permission to use the records.

The disease progresses rather rapidly, though remissions occur. In from six months to a year the process has nearly reached its height. During this time the patient emaciates, marked anemia or pernicious anemia sets in. The skin is sallow and pale, and pigmentation is seen. Diarrhea occurs at times. The bladder becomes weak and retention of urine follows; the control of the rectum is eventually lost, and the patient lies bed-ridden with paralysis and contractures of the lower limbs. The arms do not become so badly paralyzed. Death

occurs in from one-half to two years in the fatal cases. It may be prolonged to three years, and it is not unlikely that some patients can be relieved or the process checked if seen in time.

Pathology.—I have already spoken at length on this point. The disease is a toxic one, but whether due to the failure of some glandular tissue of the body to act or to infection or poison is not known. As the disease belongs to the degenerative period of life, it may fairly be supposed that in some cases

BLOOD EXAMINATION OF A TYPICAL CASE OF COMBINED SCLEROSIS.

	January, 1898.	October 1st, 1898.	October 27th, 1898.
Number of red cells per 1 c.m.m. (Thoma).....	3,000,000	2,100,000	1,840,000
Number of white cells ...	5,640	3,400	6,000
Differential Count:—			
Small lymphocytes	30 per cent.	34 percent.	38.5 per cent.
Large lymphocytes	9 per cent.	6 per cent.	13 per cent.
Polymorpho-nuclear neutrophiles	46 per cent.	53 per cent.	44.5 per cent.
Eosinophiles	3 per cent.	2 per cent.	2.5 per cent.
Abnormal Forms—			
Myelocytes	12 per cent.	5 per cent.	1.5 per cent.
Amount of hemoglobin (Fleischl)	85 per cent.	61 per cent.	52 per cent.
Specific gravity.....	1.057	1.050	1.047
Color index.....	1.4	1.5	1.4
Fibrin.....	Diminished
Blood plates.....	Diminished	Red cells large, of fair color; many polkilocytes, macrocytes, and microcytes; few normoblasts and some megablasts.	Red cells abnormally large and of good color; otherwise same as October 1st.

the blood-making organs undergo premature senility, and that the nerve centers are poisoned in consequence. In other cases less easily demonstrable metabolic perversions occur. It is not likely that the change in the spinal cord is primary.

As to the location of the trouble, the posterior columns of the spinal cord are the parts first and most involved. The process attacks these columns in their entire length, but especially involves the postero-median parts. It appears to start

in certain foci in the lower dorsal or cervical cord, and to spread thence. New foci apparently develop as the disease progresses; the lateral columns are also severely affected, and especially the crossed pyramidal tracts. The other columns are involved, but less regularly and completely. The lower dorsal and lower cervical levels of the cord are especially selected. The anterior horns are attacked and very severely, but the change here comes later. Finally, there may be actual softening in the cord, producing cavities. The blood vessels are somewhat affected with hyaline degeneration, but I am unable to say how important a factor this is. The walls are thickened, there are in places dilatations and hemorrhages, and in other places collapsed vessels. There is no evidence of inflammatory reaction. The meninges are not involved, nor are the anterior and posterior roots much affected. Examinations of the peripheral nerves (Rothmann) and of the brain have been negative.

Diagnosis.—The disease is easily distinguished from locomotor ataxia by the absence of syphilitic history, the rapid onset, the anemia, the motor weakness, the absence of ocular symptoms and of lightning pains. There is usually also at first an exaggeration of the reflexes. The steady and rapid progress of the malady is also most characteristic.

Multiple neuritis is excluded by the slow onset, the marked ataxia, the absence of muscular wasting, tenderness and pain, and the development of bladder and rectal symptoms.

I must confess that my last case presented such a marked differentiation of cutaneous sensations that I believed it to be one of syringomyelia. The necropsy showed a cavity in the cervical cord due to softening, and in a measure explained, if it did not justify, the diagnosis. The steady and rapid progress of the symptoms ought to exclude such a diagnosis.

There is little in common with the ataxic paraplegia of Gowers in this disease. This latter trouble in my opinion, can always be sifted down to some form of tabes, chronic myelitis or disseminated sclerosis. Still, some cases of tabes of irregular type suggest at first this malady, and it may require some time to enable one to make the distinction.

The existence of marked anemia, and especially of per-

nicious anemia, is, taken with ataxia, paralysis and rapid course, pathognomonic.

The essential nature of this process is a primary nerve degeneration, affecting apparently the nerve tissue first. The same poison which causes pernicious anemia does the work here, only it may affect the nervous centers without causing a true essential anemia. It seems even as if in some cases the neural change came first.

I can only say in conclusion that much light can in the future be thrown on this trouble by a study of milder clinical cases, for such, no doubt, occur. We may find it related to some definite infection which does not always hit the nerve centers with killing force. It may even be that the disseminated sclerosis of early life is a malady of the same kind, but one which does not progress on account of the youthfulness of the tissues. The tendency of neuropathology now is to lessen rather than increase the number of pathogenic processes of the nervous system, and it may well be that this combined sclerosis is only a familiar poison attacking the predisposed nervous centers of those in the declining years of life.

MENTAL DISEASES AMONG THE ARABS. Milhon. (*Annales Medico-Psychologique*, 8th Series, Vols. III. and XIV., 1896-1897.

From observations made upon the Arabs in Algiers, Milhon presents some statistics regarding their sanity. The Arabs, he claims, are as a rule, a race with sound and healthy minds, mental diseases among them being rare; and the chances for such, as compared with the French, though his statistics are made from a very small number of cases, are as 1 to 134. Alcohol in "Kiff" and syphilis seem to be the causes of such cases as are found; and as the excessive use of alcohol is becoming more common, the tendency will probably increase. The effects of the intoxication caused by Kiff are obscenity, sexual excitement, laughing, ecstasy, hallucinations of sight, and sensations as of flying. The author suggests that the religious views of the people, their tendency toward fatalism and mysticism are the causes of their more common forms of hallucination which are generally of the exalted type.

JELLIFFE.

REPORT OF A CASE OF TUMOR OF THE HYPOPHYSIS WITHOUT ACROMEGALY.*

BY CHARLES W. BURR, M.D., AND DAVID RIESMAN, M.D.

The point of interest in the case we report, or rather the point we wish to bring up for discussion, is the presence of hypophysial disease without any symptoms of acromegaly. We believe, for reasons which will appear later, that acromegaly is due to the disease of the hypophysis, and we purpose to use this case, which seemingly combats such an opinion, in an endeavor to explain the absence of acromegalic symptoms in some cases of disease of the hypophysis.

The patient, a white woman, 43 years old, was admitted to the Philadelphia Hospital on July 26th, 1897, on account of complete blindness. Her personal history was vague, and her heredity unknown. The only information that could be obtained from her was that she had been blind five years, and that her ovaries had been removed, by whom and for what reason she could not tell. Dr. Charles A. Oliver found the following ocular condition: O. D., iris dilated fully and irresponsive to light; pupil round; slight horizontal nystagmus; marked post-neuritic atrophy. O. S., iris fully dilated and irresponsive to light; disk still more atrophic from past inflammation than was the right.

She was transferred to the nervous wards with a diagnosis of brain tumor. Physical examination revealed little. She was a thin, anemic, blue-lipped woman. Her gait was neither paralytic nor ataxic, but was a little stiff. Rigidity, not very marked, was observed in both legs, especially in the right. The station was good. Both knee-jerks were increased, quick and spastic. On the right side ankle clonus was persistent and true; on the left it was abortive; that is, it ceased after three or four irregular contractions. The plantar jerk was present on both sides. Sensibility to touch and pain was normal on the legs, arms, and face. Her answers were so contradictory that it was impossible to determine the condition of the sense of smell. The heart and lungs were normal. Examination of the blood gave red corpuscles, 4,380,000; white corpuscles, 7,800; hemo-

* Read before the Philadelphia Neurological Society, October 24, 1898. For discussion on this paper see page 43.

globin, 55 per cent. The eosinophiles were much increased. The urine did not contain albumin, sugar, or casts. The mental state of the patient was much more striking than the physical. At first she was simply dull and listless, disinclined to talk, sitting hour after hour with head bowed on the chest, moving only to go to her meals and to bed, answering in monosyllables all questions put to her—in short, seeming to be most content when undisturbed. This condition continued for several months, and then changed. While still remaining ordinarily quiet and self-contained, she became at times most obscenely witty and cynically jocose. Trifling as it may seem to regard jocosity and obscenity as symptoms, they were in truth striking. Whether they were due to mere viciousness or to disease it is not possible to decide, but it is not impossible that they were due to disease, like symptoms having been noted in similar cases. During the last months of her life she became dull and stupid again, and suffered several attacks of coma, coming on suddenly and lasting several hours. She died in coma on the 19th of April, 1898.

The necropsy was made two days after death. The body was well developed and fairly muscular. Rigor mortis was very slight. Post-mortem lividity was marked. An old scar was present in the linea alba. The veins upon the chest were dilated. There was no enlargement of the extremities (hands or feet), or of the face, nor any deformity of the spinal column. There was an enormous deposit of preperitoneal fat five centimeters thick, and the omentum, mesentery, and mesocolon were also very thick with fat. The bladder was distended and projected five centimeters above the symphysis pubis. The liver was small and contracted, and failed to reach the costal margin by five centimeters. There were numerous adhesions in both iliac fossæ, and in the pelvis. The appendix was long, thin, and bound down at its tip to the pelvic wall. The right ovary could not be found. A part of the right Fallopian tube was present and adherent to a coil of small intestines that was prolapsed into the pelvis. On the left side also the ovary could not be found, and no part of the Fallopian tube remained. The round ligament was present. The uterus was adherent to the rectum on the left side. The diaphragm extended to the fourth interspace upon the left side, and to a corresponding point upon the right. There was quite a little bloody fluid in both pleural cavities. The lungs overlapped in front. A little of the thymus gland remained. There was a slight excess of clear fluid in the pericardial sac. The heart weighed 210 grams. The right auricle was slightly distended with liquid blood. The heart was small in proportion to the size of the body. The pulmonary valve was normal. The aorta was distinctly thickened. The

interior showed "willow-tree" markings, beginning sclerosis, and some yellowish patches above the sinuses of Valsalva. The mitral valve was thickened, and showed a few yellowish patches. The heart muscle was dull brown, marked by whitish striæ. The cavity of the left ventricle was small, and its wall twenty centimeters thick. Both lungs were edematous, but otherwise normal. The spleen was small, but showed no evidence of disease. The kidneys were somewhat small, and the capsules stripped with difficulty. On section the substance



FIG. I. Tumor of the hypophysis.

was congested, dark, both pyramids and cortex being a purplish red. The cortex was not diminished in thickness, its consistency was slightly increased, and the cut vessels gaped. The liver was small and hard. The calvarium was hard, ivory-like, and the diploe absent. In the middle line in front the calvarium was one-half centimeter thick. On the inner surface it was smooth, the grooves of the blood vessels were fairly well marked, and there were a few Pacchionian depressions. In front of the sella the bone was a little abraded, and the cavity itself was somewhat enlarged. The brain bulged considerably

when the dura was removed. The whole interpeduncular space anterior to the mammillary bodies was occupied by a mass about as large as a lemon, on the under surface of which the hypophysis appeared. (See Fig. I.) The tumor filled the space between the tips of the temporo-sphenoidal lobes, and was adherent to the membranes over the base. Its antero-posterior diameter was six centimeters, its lateral five and a half, and its depth three centimeters. The hypophysis (which in the plate is marked by a piece cut out; see Fig. I.) was one and a half centimeters transversely and one centimeter antero-posteriorly. The optic chiasm could not be discovered. Both third nerves were very much flattened. Anteriorly, the tumor extended a centimeter in front of the corpus callosum. On looking down upon the brain, the corpus callosum was seen to be much arched, with the convexity upward. The corpora striata on both sides, especially the left, bulged into the lateral ventricles. The tumor on section was grayish, not very hard, granular, and apparently not hemorrhagic.

The pathologic diagnosis was tumor of the base of the brain, probably originating in the infundibulum; absence of both ovaries, pelvic adhesions, beginning cirrhosis of the liver, slight interstitial nephritis, osteosclerosis of the skull, hyperplastic sclerosis of the cribriform of the ethmoid bone, and edema of the lungs.

Microscopic Examination.—Pieces of the tumor were fixed and hardened in alcohol, imbedded in celloidin, cut into thin sections, and the latter were stained with hematoxylin, hematoxylin and eosin, carmine, and Van Gieson's stain. The tumor is a spindle-celled sarcoma, with a well marked tendency on the part of the cells to an alveolar arrangement. On the surface the tumor is covered by a thin capsule of fibrocellular tissue, which sends short trabeculæ into the growth. This capsule contains a few blood vessels and a number of mast cells, mostly of small size, scattered irregularly through it. The cells of the tumor are large spindle cells with large nuclei, and run in concentric groups, forming large bundles, or whorls, of considerable size. In the center of these the cells are very near together, on account of pressure, and the nuclei appear as if piled upon one another, so that a semblance of giant cells is produced, but no true giant cells are present. The alveolar arrangement referred to is not produced, except just near the surface, by a true stroma, but merely by a vertical grouping of the spindle cells.

Another section which had been carried through what was considered at the necropsy the remains of the hypophysis shows the structure of the latter organ distinctly, and is that

from which the accompanying drawing has been made. (See Fig. II.) We find here small epithelial cells with darkly staining nuclei, closely aggregated, and here and there assuming a tubular arrangement. The tissue evidently represents the remains of the anterior lobe of the hypophysis. The connective tissue is slight in amount, but blood capillaries are abundant and well filled; colloid material is nowhere seen. Directly continuous with the glandular portion of the hypophysis is a small

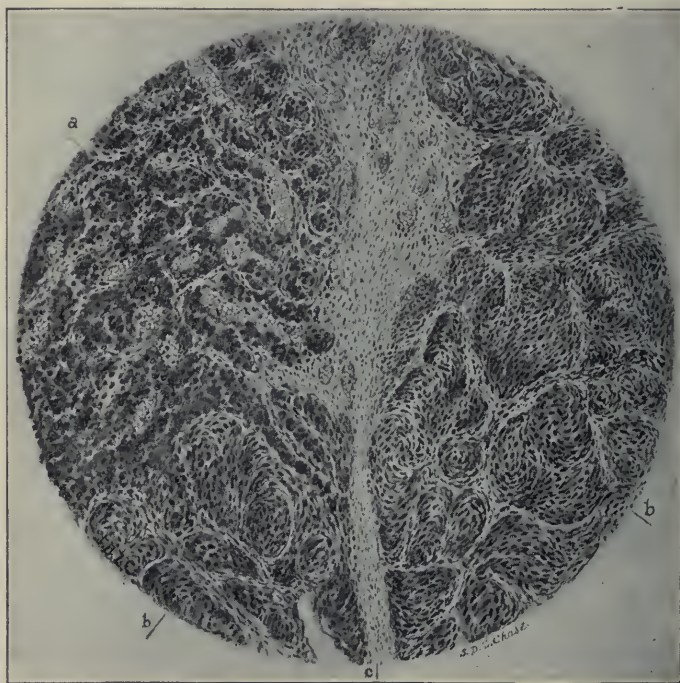


FIG. II. a—Remains of anterior lobe of hypophysis, with dilated blood vessels. b b—Spindle-cell sarcoma. c—Band of fibrous tissue. (Tissue fixed in alcohol, embedded in celloidin, and section stained with hematoxylin. Zeiss oc. 3, ob. A A.)

mass of lymphoid tissue, possibly a portion of the posterior lobe of the organ.

In one part of the section the hypophysis is sharply separated from the tumor by a broad band of fibrous tissue, as shown in the figure; elsewhere no sharp line of demarcation is observable, and in several places long, winding processes of hypophysial tissue are traceable into the new growth. The

cells in these long processes show a well marked tubular disposition, are polyhedral, and are accompanied by connective tissue and blood vessels. The processes can be traced for a long distance and are finally lost in the tumor. The tumor is practically free from degeneration.

The nervous tissue included in the section is greatly degenerated; it is loose, alveolar, and resembles in its reticular character mucoid connective tissue. The condition is well described as cribriform. The ganglion cells have lost their processes and stain indistinctly. There is no hyperplasia of the neuroglia, but in places the number of capillaries is distinctly increased.

To sum up: We have a woman whose sole subjective symptom is blindness, which came on several years before death, and who, under examination, is found to have post-neuritic atrophy in both eyes; who is usually dull and stupid, but at times very bright; who has persistent ankle clonus on one side, some rigidity in the legs and spastic knee-jerks; who has several times become comatose, and finally dies in coma.

The diagnosis of brain tumor with such a combination of symptoms is not difficult and is entirely justified. To locate the tumor is more difficult, and we found it impossible. Thus far in localization we could go; the tumor must be so situated as to encroach upon but not to destroy, nor seriously interfere with, both motor tracts, the right the more especially. Had the case been seen earlier the type of hemianopsia characteristic of hypophysial disease might have been present, but when the patient came under observation it was too late. Had it been safe to depend on her statements as to the power of smell, much light might have been thrown upon the matter, but she was entirely unreliable. Post mortem there is found a tumor involving, but not destroying, the hypophysis.

The opinion that acromegaly is due to disease of the hypophysis has rapidly gained adherents, until now it is accepted by the majority of writers. This opinion has not been formed without careful thought and wide and exhaustive discussion of both sides of the question. It is based upon the following evidence: First, that from post-mortem examination:

In Hinsdale's¹ carefully prepared list, numbering fifty-seven cases, and including all admitted by every one to be in-

¹ Boylston Prize Essay, Medicine, June to September, 1898.

stances of acromegaly, the hypophysis was diseased in all. In the cases reported by Sarbo, Friedreich and Arnold, and Bonardi, the hypophysis is said to have been normal, but there is some doubt about the correctness of the diagnosis. In William Hunter's² case, reported recently, there was vascular hypertrophy of the hypophysis with hemorrhages.

This fact, that in sixty-one cases, including even those in which there is some doubt about the diagnosis, fifty-eight showed disease of the hypophysis, proves that there must be some close relation between the two conditions. It cannot be mere accident. Were it true, as has been claimed, that disease of the hypophysis is merely a symptom of acromegaly, the lesion of the gland would always be the same, whereas it has been found to vary very much: in some cases being a so-called simple hypertrophy; in others a malignant growth.

Second, the evidence from the clinical history.

In a very large number of cases there are symptoms of tumor of the brain (headache, vertigo, optic neuritis), and in the cases which come early under medical observation, hemianopia of the type indicative of hypophysial disease is not infrequent. Furthermore, the visual symptoms increase with the increase of the bony symptoms, never beginning very late in the disease and often antedating by a considerable time the hypertrophy. The importance of this is that the visual symptoms serve in a rough way to measure the increase in the size of the hypophysis, and consequently the amount of disease.

Third, the argument from analogy with disease of the thyroid gland.

The discovery of the functions of the ductless glands is of very recent date. Not so many years ago, had any one said that the thyroid gland stood in any relation to the nutrition of the body, he would not have found any hearers. To-day every one admits that myxedema is caused by thyroidal disease. There is then in the known nature of things no reason why hypophysial disease may not cause acromegaly.

Against the view of the hypophysial origin of acromegaly there is the following evidence: First, there may be disease of the hypophysis without any signs of acromegaly. This is undeniably true. Many such cases have been reported. Weir

² Brit. Med. Jour., March 19, 1898.

Mitchell's classical case of aneurism of an anomalous artery of the circle of Willis, often quoted in this connection, has in reality little bearing, for it was reported quite a long time ago, there is no description of the condition of the extremities, and when the brain was received by him it was not in condition for any minute study. The hypophysis is not described. In none of the reported cases known to us has it been shown that the glandular part of the organ was completely destroyed; in not a few no microscopic examination was made, and in others only sufficient to determine the nature of the growth. This is the value of the case that we report. It shows that though, to the naked eye, the gland may appear greatly diseased or indeed destroyed, still it may contain much healthy glandular tissue.

Though we are ignorant whether the hypophysis contains ordinarily more glandular tissue than is needed for the proper performance of its functions, yet by analogy it is highly probable that it does. In all other glands, secretory or excretory, there is a surplus of tissue, or at least if a part be lost by accident or disease the remainder may do the same amount of work as was done by the entire organ. Nature is a spendthrift, and it is hardly fair to accuse her of niggardliness in the matter of one organ. The importance of a remnant of an organ is well shown in the thyroid. Operative myxedema does not follow the partial removal of the thyroid gland, even if the part left is very small, but does follow when the entire organ is removed. The same thing may be true of the hypophysis: namely, that a small amount of glandular tissue may do the work of the whole, or at least may be sufficient to inhibit excessive growth in the bony extremities. There may be, and probably are, other functions as yet undiscovered, requiring a greater amount of gland for their performance.

Second, the existence of acromegaly without visual symptoms. Dercum has reported two such cases, and there have been not a few others. If these cases come to necropsy and the hypophysis is found normal, their value will be greatly increased.

Third, there is not a single ductless gland in the body that has not been found diseased, and it would be as proper to say that disease of any one of them caused acromegaly as of any

other. It is true that all or almost all have been found diseased, but not any one except the hypophysis in any large number of cases. We cannot say of any other one that it was pathologic in fifty-eight out of sixty-one necropsies. Furthermore, each of them is frequently diseased without any sign of acromegaly.

Fourth, the evidence from experimental destruction of the gland. The hypophysis has been destroyed without causing acromegaly (Marinesco, Varsale, Succhi.) The acceptance of this evidence assumes the identity of function of the gland in man and dogs. This identity is assumed, not proven. We know little of the function of the gland in man, and less concerning it in other animals. The same evidence would prove that myxedema has no relation with the thyroid gland, for its removal in dogs causes entirely different, non-myxedemoid symptoms. Furthermore, acromegaly may be due to a perversion of function and not to its abolition.

Fifth, the failure of the therapeutic test. No patient has been cured by the use of hypophysial extract. This has manifestly little weight, and no one has laid great stress upon it.

Balancing all the evidence on both sides of the question, we feel justified in believing that acromegaly is caused by disease of the hypophysis, and that for its production the lesion must be complete, i. e., must affect the entire glandular structure.

REFLECTIONS ON THE NOSOLOGY OF THE SO-CALLED FUNCTIONAL DISEASES.*

BY JOSEPH COLLINS, M.D., AND JOSEPH FRAENKEL, M.D.

The authors endeavored to prove that the various so-called functional nervous disorders were primarily conditioned through the sympathetic system, and were the result of a trophic change.

DISCUSSION.

Dr. F. X. Dercum said that there was not a scintilla of evidence that the sympathetic system played the extensive role assigned to it by Drs. Collins and Fraenkel. Beginning with neurasthenia as an example, there are no grounds whatever for classifying neurasthenia among the affections of the sympathetic nerve. Neurasthenia is nothing more or less than a fatigue neurosis, and its symptoms can best be explained on the ground of a fatigue of the organism as a whole. Certainly the psychic symptoms of fatigue must be referred to the cerebrum. In what other way are we to explain the loss of power for sustained attention, the loss of spontaneity of thought, the loss of power for sustained intellectual effort? Similarly, the general motor weakness of neurasthenia must be referred to the cerebro-spinal system as a whole. All of the symptoms of this disease are symptoms of general nervous weakness and nervous irritability and not of vasomotor or sympathetic disturbances. The only indication of any role played by the sympathetic is in the vasomotor disturbances. Vasomotor weakness and irritability were long ago proven by Anjel by means of the plethysmograph, but these symptoms are merely part of a group. Much of the confusion with regard to neurasthenia arises from the fact that in neurasthenia there are two sets of symptoms—primary and secondary; the first being the essential symptoms of fatigue; the others, secondary outgrowths of these. Charcot was the first to recognize that a difference in value obtains among the various symptoms of neurasthenia. The fatigue symptoms, for instance, are readiness of exhaustion, inability for sustained intellectual or physical effort, headache, backache, limb-ache, etc., but to these symptoms are added others, such as tinnitus aurium, giddiness, throbbing and allied sensations

*Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

which are, in all probability, to be referred to vasomotor disturbances and to rise and fall of blood pressure, but they are all of them secondary in value and not primary, and in many cases are altogether absent.

Again, as regards uric acid, Dr. Dercum said that he was one of those who did not accept the view that uric acid was a result of insufficient oxidation of the tissues. Horbaczewsky showed some years ago that protonuclein can be converted into uric acid in the laboratory. If this is true, an excess of uric acid simply means an excess of tissue waste.

As regards hysteria, Dr. Dercum said that the symptoms of the disease were such as to refer us beyond all possible doubt, not only to the cerebrospinal system, but especially to the cortex. In what other way are we to explain, for instance, the symptoms of hemianesthesia, or such a symptom as segmental anesthesia? A segmental anesthesia cannot be referred to a nerve distribution; it cannot be referred to the sensory representation in the spinal cord; it must be referred to the cortex, and there are other facts which give us every reason to believe that the representation of the limbs in the cortex is by segments. To attempt to explain hysteria by means of the sympathetic system is equivalent to offering no explanation at all.

Dr. Charles K. Mills said that many years ago he had written one of the old-time reviews of a book of several hundred pages for the *American Journal of the Medical Sciences*. This book was written by a French physician, Trumet-de Fontarce, who attributed almost all the diseases referred to by Dr. Collins and Dr. Fraenkel, except perhaps acromegaly and one or two other recent affections, to the sympathetic nervous system. Dr. Mills said that if Dr. Collins and Dr. Fraenkel had not had access to that book they would find it an interesting work in connection with the subject under discussion.

Dr. Mills thought that Dr. Collins and Dr. Fraenkel had made the common mistake of attributing to a certain portion of the nervous system diseases which simply manifest themselves through this portion. They had done little more than assert that certain diseases were traceable to the sympathetic nervous system. To a certain extent the paper was a step backwards; it was a reversion to the old method of looking upon the system of gangliated nerves as a distinct system, which it is not. We have quite as much evidence in favor of designating hysteria a cerebral or psychic disease as we have in favor of designating it a disease of the sympathetic nervous system.

Dr. B. Sachs did not know whether he was one of those who enjoyed that "immunity to scientific evidence" to which the paper referred. He thought that Dr. Collins and Dr. Fraenkel had done good work in calling attention to the sympa-

thetic nervous system as a possible factor in functional diseases, but he did not think it was wise to draw too general conclusions from this. Still, we are not in entire ignorance of the functions of the sympathetic nervous system. We know that certain pupillary and visceral symptoms, as well as certain vasomotor and trophic symptoms, have their origin there. He did not think the writers of the paper would be able to prove that such symptoms occurred more frequently in hysteria or neurasthenia or acromegaly than they do in diseases which we positively know are of cerebrospinal origin.

Dr. William Osler regarded the bringing forward of this old, defunct theory as a very retrograde step. Still, he said, it only goes to show that we think the same thoughts our fathers thought.

Dr. W. L. Worcester believed the paper was open to objection on theoretical grounds. It was generally acknowledged by students of evolutionary processes that those structures which were latest developed were the most liable to disorder. The sympathetic nervous system is a more primitive portion of the nervous system than the cerebrospinal system; it is mainly concerned with the regulation of the circulation and secretion. When we have to deal with a disorder of the cerebrospinal system, he thought it less reasonable that we should search for the cause of such disorder in the fundamental and simple sympathetic system than in its own and less primitive structure.

Dr. Van Gieson said that while he agreed with Dr. Osler that many of the statements contained in the paper of Dr. Collins and Dr. Fraenkel had been said before, he thought it was necessary, from time to time, to say them over again. While it was very true that the higher parts of the nervous system were evolved last, it was also true that they are most dependent upon a continuous food supply. The authors of this paper had attempted to show that in certain diseases there are changes in the higher spheres of the nervous system produced by functional or organic changes in the sympathetic system, which have deprived those higher centers of their food supply.

Dr. J. J. Putnam did not regard the sympathetic system as an essential factor in the causation of disease, but looked upon it as a very important factor in the maintenance of certain diseased conditions. In Graves' disease, for example, the onset may be due to a fright, which is in the first instance purely psychic; the symptoms it produces are taken up by the sympathetic system, and if this system becomes seriously involved those symptoms are not easily recovered from, and form an essential part of the disorder.

Dr. Collins said he was fully prepared for the criticisms that had been made by the various speakers on the paper which

he and Dr. Fraenkel had just presented. He felt moderately confident that the theory which they had advanced did not rest upon mere assertions, but that it was supported by tangible scientific evidence. If he were as capable as Dr. Dercum of making lucid his thoughts he could wish for no better words to maintain the position that he and Dr. Fraenkel had taken, relative to neurasthenia in its relation to the sympathetic nervous system, than those employed by that speaker. They did not claim in their paper that neurasthenia was a disease of the gangliated cords, of the communicating fibers, of the terminal ramifications, or of any other part of the sympathetic system; on the contrary, they distinctly stated their belief that the symptoms of neurasthenia were primarily conditioned by the sympathetic system, and that they were the result of a nutritional change. Dr. Dercum said he believed that certain symptoms of neurasthenia were due to changes in the blood pressure, and to that extent, at least, he agreed with the authors of the paper. How are these variations in blood pressure brought about? Does the cerebrospinal system send any filaments to the blood vessels?

In reply to Dr. Sachs, the speaker said he had recently made some experimental researches, together with Dr. Onuf, on the localization of the sympathetic nerve in the brain and spinal cord. The result of these researches, which were to be read later by Dr. Onuf, easily explained that the implication of the sympathetic system was responsible for the symptoms of sympathetic disorder occurring in the course of cerebrospinal disease.

Dr. Collins said they did not wish to contend that the diseases mentioned were diseases of the sympathetic system. They had merely advanced the claim that these diseases are more legitimately interpreted by positing disorder in function or disease of the sympathetic nervous system, as the *sine qua non* of their existence, than by any other explanation, be it chemical, reflex or anatomical. They maintained that the diseases were nutritional diseases, and that the sympathetic nervous system controls nutrition from beginning to end. Dr. Osler had pronounced their theory a retrograde one. He would ask Dr. Osler to supply him with a better one.

Dr. Collins said that while their views on rheumatism and gout might be termed retrograde, *chacun son gout*; he considered them more satisfactory than any that had been brought forward, even though the French author to whom Dr. Mills referred had written so voluminously and unconvincingly about them.

Society Reports.

THE NEW YORK NEUROLOGICAL SOCIETY.

November 1st, 1898.

The President, Dr. Frederick Peterson, in the chair.

A DOUBTFUL CASE OF INFANTILE SCURVY.

Dr. Joseph Collins presented for diagnosis the case of a girl of three years, born of healthy parents, after an easy and normal labor. About January 30th the right eye of the patient was slightly closed, the eyeball was somewhat shrunken, and a discoloration was present around the eye, as though a blow had been received there. After two or three weeks the patient did not use the right leg and the right arm. There seemed to be considerable tenderness over the right upper extremity, and particularly over the brachial plexus. This disappeared after four or five weeks, and then the ocular symptoms first observed returned. This was her condition when first seen by Dr. Collins on April 30th. The girl was then rather bright, and not as irritable as she is at present. The formation of swellings on the head, like those of angioneurotic edema, was noticed at that time. Although the patient was put on restorative medicines during the summer, by fall her condition had become worse. At present her temperature is almost continuously about 103° F.; she is very irritable, and occasionally the neck becomes stiff. Some of the swellings referred to are now on the head. Ophthalmoscopic examinations had given negative results. The pupils are normal in size and in reaction to light.

Dr. Mary Putnam Jacobi said that the swellings were so much like those from subperiosteal hemorrhage, that she was inclined to regard the case as one of infantile scurvy.

Dr. George W. Jacoby said that the case made the same impression upon him, although the history was negative as regards the occurrence of hemorrhages from the gums.

Dr. Peterson took the same view.

Dr. W. M. Leszynsky said that he had met with several children having subperiosteal effusions and thickenings without any hemorrhages from the gums. These cases recovered. He did not see how a positive diagnosis could be made unless the child were kept for a considerable time under constant observation in a hospital.

Dr. Collins replied that the diagnosis of scurvy had suggested itself long ago, but antiscorbutic diet and treatment had been tried without any benefit. The urine and blood had been examined with negative results.

A CASE OF SUPERIOR TABES.

Dr. Onuf presented a colored man having a personal history of little clinical importance. The first symptom of his present disease appeared in 1895, and consisted in a feeling of cold over both patellæ. About a year later he was struck in the right popliteal region, and for two or three days afterward the right limb felt stiff. In April, 1896, his vision became blurred. He states now that his lips feel stiff and his tongue dry; that there is a throbbing sensation in the right ear; and that his mouth often opens without his noticing it. Sexual desire has markedly diminished, but he has no bladder disturbance. Examination shows certain peculiar contractions of the facial muscles. The left pupil is wider than the right, but both of them are abnormally narrow. Accommodation is good, and the ocular muscles move naturally with the exception of an occasional slight nystagmus. The sensation of the face is markedly impaired, and the analgesia extends also to the mucous membrane of the mouth. The mouth on being opened widely is twisted to one side. The gait is disturbed, but it can hardly be called ataxic. The patient stands steadily with the feet together and the eyes closed. The knee-jerks are present on both sides. Some ataxia is noticed in both upper extremities. The visual fields on both sides are somewhat contracted, and both optic nerves are atrophied.

Dr. George W. Jacoby remarked that he did not think these cases of superior tabes were very rare.

ATYPICAL PROGRESSIVE MUSCULAR ATROPHY.

Dr. Pearce Bailey presented a case of ophthalmoplegia externa, with symmetrical atrophies in the extremities, which he regarded as one of atypical progressive muscular atrophy. The patient, a man of twenty-six years, had been well up to two years ago. He had been athletic, and was not given to any excesses. No predisposing or exciting causes of nervous disease could be discovered in his ancestral or personal record. The first symptom of the present illness occurred in June, 1896. It consisted of drooping of the left eyelid and rotation outward of the left eyeball, unaccompanied by pain. It was rapid in its onset, so that within three or four days of its first appearance the ptosis was pronounced and diplopia was present. This condition in the left eye remained, with some intervals of improvement, until a few months ago, when the right eye became affected in a similar way. As the muscles of the right eyeball became impaired those of the left regained much of their power, so that at present the defects on the right side are much more

conspicuous than those on the left. The diplopia disappeared and then returned, and is now present.

To this alternating ophthalmoplegia, weakness in the extremities was added about a year ago. In the upper extremities it was confined to the triceps of both sides, and in the lower extremities to both anterior tibial groups. In these latter it was first manifested by a slight dragging of the feet in walking. Occasional difficulty in holding the water has been noticed since the beginning of the trouble: i. e., the urine must at times be voided at short intervals, although true incontinence was never present. The general nervous tone of the patient is also lowered. From being an active athletic young man he has become more or less of an invalid. He has lost fifteen pounds. He tires easily; he is easily startled; he becomes nervous and apprehensive at slight causes; his sleep is disturbed. Large doses of iodide have been taken for long periods without appreciable benefit.

Examination shows a young man of slight frame, but well proportioned and of good general muscular development. No traces of disease exist in the vegetative organs. Dr. W. A. Holden reports that in the right eye there is marked paresis of all the extrinsic muscles supplied by the third nerve, and in the left eye a less degree of paresis of the corresponding muscles (the levator and internal rectus muscles being but slightly affected). Each external rectus is somewhat weak, extreme abduction being accomplished only spasmodically. Pupillary reaction, accommodation, acuteness of vision, fields and the interior of the eyes, are normal. The other cranial nerves appear to be normal. Weakness and atrophy of both triceps and of both anterior tibial groups are found in the extremities. The loss of power in the triceps muscles is the more pronounced, although it causes the patient no serious disability. These muscles are noticeably atrophied, and occasionally are the seat of fibrillary twitchings. It is more difficult to demonstrate atrophy in the muscles on the front of the leg. The weakness in the latter, however, becomes apparent occasionally when the patient walks, and always when he tries to lift the toes from the floor. Changes in electrical excitability of the same character in all the affected muscles are found, although these are most pronounced in the triceps muscles. The changes consist in a diminished response to faradism. To galvanism there is no reversal of reaction, but the contractions in places are distinctly vermicular. The knee-jerk on the right side is absent; on the left, a very deficient knee-jerk may be obtained by reinforcement. This is practically the full symptomatic category. There are no atrophies elsewhere, no spasmodic contractures or contractions, no disturbances of cutaneous sensibility.

Dr. C. L. Dana said regarding the prognosis of the disease, that the duration of life was sometimes considerable. He had seen a physician in whom a progressive muscular atrophy had developed in early manhood, and had then ceased to progress, so that he had been able to practise medicine for twenty years. This man had checked the progress of the disease by going to bed and keeping quiet. Dr. Dana said that he had seen other cases of this kind, and at present had under observation a lady of fifty years in whom the atrophy had been held in check for twenty years. The case just presented might be a dystrophy, but this did not seem probable because of the optic atrophy.

Dr. Peterson said that he had examined this case, and while he inclined to the diagnosis given, the case certainly presented some unusual features. In the first place, it was unusual for progressive muscular atrophy to exhibit itself symmetrically in the different extremities. One physician had diagnosed the case as syphilitic; if this were true possibly a syphilitic neuritis was present.

Dr. Dana said that when progressive muscular atrophy began with ophthalmoplegias and then proceeded downward it was likely to be symmetrical. He recalled one case in particular, giving a history almost precisely the same as that of the case just presented.

REPORT OF A CASE OF TUMOR OF THE SPINAL CORD.

Dr. Joseph Collins presented this report. The patient was an unmarried negress, twenty-three years of age. She said that on August 29, 1898—two weeks before admission to the hospital—she had noticed a feeling of numbness and loss of power in both legs. The loss of power increased rapidly, and in five days she was completely paraplegic. Retention of urine, she asserted, began on August 29th, and along with this there was incontinence of feces. She next experienced a sensation of burning and formication in the limbs, but she did not complain of pain—a point of special interest. Her family history was incomplete, but not noteworthy. Early in 1896 she had sought relief from amenorrhea, and had been subjected to some operation the nature of which she did not know.

On inspection, emaciation was extreme, and the suffering seemed to be great. A large bed sore was found over the sacrum. She appeared to be unable to make any voluntary movements of the lower limbs. Sensibility to touch, temperature, and pain was completely lost in the lower extremities, and nearly as high on the trunk as the crest of the ilium. Local elevation of temperature of 1.5° F. was noticed over the abdomen below and immediately around the umbilicus. Palpation of the abdomen showed an immovable nodular mass on the left side, extending towards the liver on the right side. The back was fixed rigidly, and the lumbar region was arched. Pressure over the ninth dorsal vertebra elicited pain. No sensory disturbances were noticed in the upper portion of the body. She died on September 15th.

At the autopsy a reddish soft mass was seen lying over the lateral aspects of the cord from the twelfth dorsal to the fourth lumbar vertebra. The mass was a dural tumor growing through the dura and implicating the pia. The growth was connected with an abdominal neoplasm. The uterus and adnexa had been removed. The firm immovable mass was apparently retroperitoneal, and attached to the bodies of the vertebrae from the tenth dorsal to the second lumbar vertebra. The mesentery in the vicinity contained many small, whitish nodules. Microscopical examination showed both the dural and the abdominal tumors to be round cell sarcomata.

The noteworthy features were: (1) The sudden onset; (2) the simultaneous and complete overthrow of the functions of the bladder and bowel; (3) paraplegia dolorosa; (4) localized pain in the back; (5) absence of radiating neuralgic pain in the extremities; (6) the early occurrence of trophic symptoms in the shape of a bedsore.

The records at the Roosevelt Hospital showed that the woman had been admitted in April, 1896, and that she stated that she had never menstruated, and had noticed during the previous year a gradual increase in the size of the abdomen. She had an infantile uterus. On April 4th, complete abdominal hysterectomy was performed. There was a nodular tumor of the left ovary; the right tube was normal and extremely infantile, and the uterus appeared as a slim cord in the broad ligament. The microscope showed the tumor to be an endothelioma. The diagnosis in the hospital was sarcoma of the uterus and infantile uterus.

Dr. Charles L. Dana read a paper on "The Subacute Combined Scleroses of the Spinal Cord, and Their Relation to Anemia." (See page 1.)

Dr. W. B. Noyes said that the case just reported corresponded closely to one coming under his observation recently at the Columbus Hospital in the person of an Italian of thirty years. He presented at first the ataxia of a spastic paraplegia, with comparatively little sensory disturbance. Anemia was marked, and an examination of the blood showed a diminution in the number of the red corpuscles and of the hemoglobin, but no megaloblasts. The patient recovered rather rapidly from his paraplegia under the administration of iron. His eyes were examined by Drs. Callan and Holden, who found the retina extremely hydremic. There was no albuminuria, but there was marked hydremia. Although the man was almost completely paraplegic on admission, in two weeks he was able to walk around, and ultimately completely recovered from his paraplegia.

Dr. L. Stieglitz remarked that the changes in the cord did not seem to him essentially different from those reported by Nonne. He had never seen a case develop into tabes.

Dr. Carlin Philips said that, aside from the clinical course, there were certain changes in the bone marrow and in the liver which were more or less pathognomonic. He had found undoubted degeneration of the cells of the cortex by Nissl's stain, not only in the motor zone, but in the cuneus, angular gyrus and in some of the frontal convolutions. Our conception of pernicious anemia seemed to be somewhat confused; the general tendency was not to consider it a distinct disease.

Dr. Fraenkel said that he now had under observation three women, all of whom presented symptoms explicable on the theory of combined sclerosis. There was no decided anemia, although there were vascular changes. The pernicious anemias certainly lead frequently to thromboses, as do also sclerotic conditions, and hence it seemed to him better to look upon the spinal-cord conditions as softenings resulting from such vascular lesions. The paper was a valuable aid to the diagnosis of those obscure spinal-cord lesions so commonly met with in old people—those cases formerly classified in a general way as ataxic paraplegias, without any clear idea of their pathology.

Dr. Terriberry said that he had at present under observation a woman of about fifty years, who presented symptoms of ataxia, increased by attention. She was well nourished and had a good family history. Nothing else could be discovered except a slight increase in the knee-jerk and an underlying gouty condition. So far as he knew her condition had existed for a dozen years, and certainly the phenomena mentioned had changed very little in the three years she had been under his care. Under the use of the iodide of strontium her condition had improved. He considered the case as fundamentally one of gout.

Dr. Dana, in closing the discussion, said that he wished to insist especially upon the fact that there is a group of cases which can be recognized clinically, and underlying which is a pathological condition described in his paper. Sometimes these were cases of pernicious anemia, but in the majority of instances they were not. There was often a strong secondary anemia, but even this was not infrequently absent. The changes that he had observed were much more pronounced than those of pernicious anemia. Dr. Burr had stated that in pernicious anemia there was no vascular disease. He had himself examined the cortical cells in severe secondary anemia, and had noted marked pigmentary degeneration. One very striking characteristic of this malady is its rapid course. Some cases of ataxic paraplegia undoubtedly come on suddenly, usually as a result of a thrombosis or rupture of a vessel in the cord, but they can be excluded by the mode of onset and by the duration.

THE PHILADELPHIA NEUROLOGICAL SOCIETY.

October 24, 1898.

The President, Dr. F. X. Dercum, in the chair.

A CASE OF TETANY.

Dr. Elizabeth R. Bundy presented a case of tetany from Dr. Spiller's clinic. The patient was a man twenty-two years of age, a native of Austria, who had been eighteen years in this country. He had been seized while on the street with violent spasms. These were frequent, beginning in the feet or hands, and attacking in turn nearly all the muscles of the extremities, as well as those of the head, chest and abdomen. The fingers were flexed upon the palm and the thumb upon the fingers, making a fist rather than the more usual "obstetrical hand." The forearms were flexed and the arms adducted, and toward the end of the spasm the claw-hand was produced for a moment or two. The toes were flexed, the feet were in the position of talipes equinovarus, and the entire lower extremity on each side was flexed and adducted. The face and forehead were also plainly involved in the spasms, which were so frequent and painful that an electrical examination was not made at this time. The electrical examination made later did not give abnormal reactions. The sensibility of the nerves was increased and slight pressure over the ulnar or external popliteal nerve caused numbness and tingling in the peripheral distributions. Trousseau's sign was easily obtained, but Chvostek's sign could not be elicited.

The diagnosis was based upon the character of the spasms, inasmuch as they were bilateral, intermittent, painful, beginning in the extremities and developing toward the trunk; on increased sensitiveness of the nerve trunks to pressure; on the presence of Trousseau's sign, which could be obtained at least two weeks after the spasms had ceased; on the presence of subjective sensory symptoms, namely, tingling of the skin and gastric irritability preceding the onset of the attacks for a day or two.

The patient gave a history of previous attacks during the past three or four years, occurring always in mild weather and lasting from three days to two weeks. No other member of the family seemed to be similarly affected.

Dr. William G. Spiller said he had seen quite a large number

of cases of tetany in Vienna and had had the opportunity of observing carefully the case just described. He regarded it as undoubtedly one of tetany. Only two other conditions could be considered: hysteria and simulation. The occurrence of this disease in a person originally from Vienna, although he had been in this country eighteen years, the occurrence in a young male, and only in warm weather, were interesting statements. The spasms were somewhat atypical, but this type has been observed before. The involvement of the lower extremities and of the whole body is well known. The absence of increased irritability of the facial nerve and of the other motor nerves was uncommon, although the irritability of the sensory nerves was exaggerated.

Dr. Spiller thought that simulation need not be considered. The boy had never seen a case of tetany and knew nothing of the disease. Trousseau's sign is characteristic of tetany and was so marked in this case, that pressure over the large vessels and nerves of the arm caused a violent spasm of the muscles of the hand. The boy had stated that a fracture of a phalanx of one of the toes had been caused on one occasion by the attempt to straighten the toe while it was in spasm.

Dr. Joseph Sailer said he had seen a patient two years ago with tetany, who also had come from Vienna. The patient was a girl ten years of age and had been in this country two years. The symptoms were exceedingly mild in character. In the morning, she would have a slight cramp in the hand, soon disappearing and not recurring during the day. The characteristic signs were present, but slight. The diagnosis was confirmed by the electrical examination, which showed that the susceptibility of the nerves was greatly exalted. The patient passed from observation. She was one of a large family and none of the other members had ever shown symptoms of the disease.

Dr. James C. Wilson added another unpublished case. His patient was a young Frenchman thirty-two or thirty-three years of age, who after a series of business reverses went into ill health with gastro-intestinal catarrh and chronic diarrhea, and in the latter period of his illness developed well marked tetany. The symptoms of tetany continued for about two weeks, at the end of which time the patient died, chiefly in consequence of the intestinal trouble, but in part also from the exhaustion from the recurring attacks of spasm.

Dr. David Riesman said that about two years ago he had seen a case of tetany with Dr. Gordon Ross. The patient was a boy probably ten years old, a native of the United States. At first he appeared to be suffering from tetanus. There was, however, no reflex motor excitability, but most of the signs of tetany could be obtained. There was a marked hyper-extension of the foot with flexion of the great toe. The thyroid gland was believed to have been given without benefit. An attempt was made to find ptomaines in the urine, as it was thought that the condition was due to gastro-intestinal intoxication, but on account of decomposition of the urine the search was discontinued.

With regard to tetany in dogs, Dr. Riesman said he had seen it a number of times. Dr. Carter and he in some experiments found that tetany usually developed within twenty-four hours after the removal of the thyroid gland. It has been held that this result could be prevented by not giving a diet rich in proteids. They attempted this in one case, but without result. It may be that the age of the dog has some influence. It was the experience of one of the pupils of Kocher that the age of the animal was a factor in the development of tetany.

A CASE OF ATAXIA LIMITED TO THE RIGHT ARM.

Dr. F. X. Dercum presented a woman of twenty-six years, who began to have numbness in the fingers and thumb of the right hand three months previously. Subsequently the fingers and hand became stiff, and she dropped various articles which she attempted to hold. A sense of stiffness and of dead weight subsequently and gradually spread up the forearm and arm as far as the shoulder. The right arm could be extended from the shoulder joint and moved freely in all directions, but the movements were awkward. The arm was readily flexed and extended at the elbow, but on attempting forcible extension of the biceps the arm was found to be less strong than its fellow. Movements of the wrist were performed freely in all directions; the extensors, however, were weaker than those of the opposite side. The grip of the right hand registered 22; that of the left hand 35. Movements of the fingers were executed freely, but were decidedly ataxic. The movements of the entire limb were ataxic. The tactile and temperature senses in the right limb were preserved, but the pain sense was slightly diminished over the forearm and fingers. The stereognostic sense in the right hand was lost. The patient complained of stiffness of the neck, and the movements of the neck were slightly restricted in all directions. More recently the left arm had begun to be a little weak. The sway was slightly exaggerated, but the gait was normal. The right knee-jerk was increased, and weak ankle clonus was present on the right side.

Note.—In a short time paralysis developed in all the extremities. The patient died about three weeks after she had been presented before the society. At the necropsy a tumor was found growing from the bone on the right side of the foramen magnum, and greatly compressing the medulla oblongata.

Dr. Charles K. Mills thought that this case was possibly cerebral and due to a focal lesion in the posteroparietal region of the cortex, or more probably to a lesion more deeply situated, but related in some way to the tracts which terminate in this region of the cortex or in the thalamus. It could hardly be a purely hysterical case, as many of the points developed were opposed to this view.

Dr. Charles W. Burr said that two years ago he had reported the case of a man who many years previously, after an accident to the head, had been palsied on the left side. He recovered from this palsy completely. After he was able to walk he chanced to put his hand into his pocket and found that he could not recognize what he took into his hand. He was told that this was a matter of no importance and that he would soon recover. That was fifteen years ago, and the condition still remains. When Dr. Burr saw the man there was no objective sign of disease except the scar. The patient was able to feel the lightest touch, but with his eyes shut he was

unable to tell what position the arm or fingers were in, and could not recognize objects by touch.

Dr. Burr thought that the reason we are able to tell the shape of objects felt, is that we know the position that the hand must assume in order to grasp the object. In these cases the patients do not know what position the hands are in and hence do not recognize the shape of the object.

Dr. A. A. Eshner thought that the inability of Dr. Dercum's patient to determine the character of an object placed in her hand might be explained by a disturbance of peripheral sensory localization. If this girl could feel the object at each point where it touched her hand, she could probably determine its outline and arrive at a conclusion in regard to its form and size, but not necessarily as to its weight or its temperature.

Dr. F. X. Dercum said that if it were not for the marked rigidity of the neck and of the slight ataxia of the *left* arm, he would almost, without hesitation, have pronounced this to be a cerebral case. This rigidity might be due to a complicating rheumatic affection, or it might be due to a pachymeningitis. The iodides and mercurials had been used in this case and were well tolerated. This was very suggestive, of course, as pointing to possible multiple lesion.

A CASE OF BRAIN TUMOR.

Dr. J. L. Nicholson gave the history of a man who had had symptoms of brain tumor. In October, 1896, the patient fell and sustained a slight injury to his head. In the following December he fell down a stairway and injured the occipital region. He was slightly stunned for a few minutes. On February 14, 1897, while traveling in a railroad train, he felt a pain in his right leg and right arm, and became unconscious. On March 10, 1897, he had another attack of unconsciousness while on a train. On April 7 he had convulsions limited to the muscles of the right side of the body, lasting about three minutes, and he is said not to have lost consciousness at this time. The convulsions began in the right foot and leg, and then involved the trunk, arm, neck and face. The muscles of the right side, when the patient was examined, were found to be much weaker than those of the left. Ankle clonus and exaggerated reflexes were found in the limbs of the right side. No marked objective disturbance of sensation was noted, although the right leg and arm felt numb. No abnormal condition of the eye was found at this time, but one year later Dr. Risley noted optic atrophy of the outer side of each disk and signs of hemorrhages. The patient's memory was good until late in the disease, except shortly after one of his attacks. He had many right-sided convulsive attacks, and suffered from nausea and vomiting.

An autopsy was obtained August 28, 1898. A tumor was found slightly raised above the surrounding cortex, measuring $1\frac{1}{2}$ inches anteroposteriorly and $1\frac{1}{4}$ inches vertically. It was

situated at the upper portion of the fissure of Rolando on the left side of the brain, and dipped down into the longitudinal fissure $\frac{3}{4}$ of an inch. The tumor was not sharply defined from the surrounding cortex. Microscopically it was a round cell sarcoma.

Dr. Charles K. Mills said he had seen the man about a year and a half before the patient's death and had advised very strongly in favor of operation. He regretted that this advice had not been followed, as the case appeared to be an excellent one for operation.

Dr. William G. Spiller had seen this patient with Dr. Mills. Both Dr. Mills and he had urged an operation. The man had had many epileptic attacks, always limited to the right side, optic atrophy, weakness of the right side of the body, nausea, vomiting, headache and disturbance of the so-called muscular sense in the right upper and lower limbs. Dr. Spiller believed that the tumor was not very deep in the cortex. The epileptiform convulsions indicated a rather superficial lesion, and the paresis rather than paralysis, looked as though the growth had not penetrated very far into the motor cortex. The condition of the muscular sense, or sense of position, led him to believe that the parietal lobe was involved. He said that the supposed location of the muscular sense in the parietal lobe is of great interest, although we do not know that this view is correct. As the man could read it was not probable that the angular gyrus of the left side was invaded, provided we accept this gyrus as the center of word hearing.

Dr. Spiller reported a case which Dr. Lloyd and he had observed in the clinic of the former at the Philadelphia Hospital. A man, well advanced in years, complained during the night preceding his attack, of fatigue, headache and inability to sleep. In the morning he fell to the floor on attempting to rise from his bed, but was not unconscious. When he was found he had paresis of the left limbs and of the left side of the face and tongue. The left knee-jerk was exaggerated, and the left lower limb was rigid. Later the patient could raise his left upper limb above his head, but every movement of this limb was ataxic in an extreme degree, though the limb was not paralyzed. The mental condition of the patient prevented an examination of the condition of sensation.

At the necropsy, a hemorrhage was found in the right parietal lobe. The hemorrhagic area was about two centimeters in diameter and extended inward in the form of a cone to the lateral ventricle, having its base in the cortex. It was situated about four centimeters from the longitudinal fissure, and about two or three centimeters behind the Rolandic fissure. When Dr. Spiller saw the brain, it had been cut into frontal sections about a centimeter apart, and although the injury to the cerebral tissue prevented an exact determination of the location of the hemorrhage, it was evidently in, or very close to, the supramarginal gyrus.

Dr. Spiller thought that this case, together with the statements of v. Monakow and others, relative to the muscular sense, rendered the diagnosis of a cortical lesion in Dr. Dercum's case of ataxia in one arm not unreasonable.

Dr. Charles W. Burr and Dr. David Riesman reported a case of tumor of the brain, by which they attempted to show that acromegaly is due to disease of the pituitary body. (See p. 20.)

Dr. F. X. Dercum thought that the conclusions of Dr. Burr and Dr. Riesman would have been more completely established if they had reported a case in which the pituitary substance was all destroyed and there was no acromegaly. He could not see the close relation which this case bore to acromegaly, nor how it strengthened the view that disease of the pituitary body is the cause of acromegaly. It seemed to him further that the assumption that the pituitary body has different functions in different animals is a dangerous one. The pituitary body is a constant and fundamental structure in all vertebrates and presumably should possess equivalent values in all of them.

Dr. David Riesman said he might be able to answer one of the points raised by Dr. Dercum, namely, the failure to produce acromegaly in animals by the removal of the pituitary body. We cannot always apply the results of experiments on animal to man. For example, it is impossible to produce myxedema in dogs by the removal of the thyroid gland; we can produce tetany, but not myxedema, yet it will not be disputed that in man the absence or degeneration of the thyroid gland leads to myxedematous conditions.

What they attempted to show in their paper was merely this: that the cases of tumor of the pituitary body with no clinical signs of acromegaly cannot be directly used to controvert the view that disease of the pituitary body is the cause of acromegaly. If these cases had been examined with sufficient care to show that all of the pituitary body was destroyed and absent, then they might prove something. They might show that the total destruction of the pituitary body need not be followed by acromegaly and, as a corollary, that disease of the pituitary body was not the causative lesion. Dr. Burr and Dr. Riesman had shown that in their case some of the pituitary body had remained and they had inferred that this was sufficient to carry on the functions of the gland. It might be said that the remaining part was too small to have any important function. We know, however, that the smallest piece of pancreas is sufficient to prevent glycosuria in animals, and that a very minute portion of the thyroid gland is sufficient to hold in abeyance the symptoms that follow the complete removal of that organ.

If we take the ground that acromegaly is due to the destruction of the pituitary body, some light is thrown on the function of the gland. The theory of its function that had suggested itself to Dr. Riesman might be formulated as follows: All tissues have a tendency to grow until a certain period when most of them cease in obedience, it has hitherto been said, to the law of growth. This law of growth is a vague term. The tissues grow by reason of some substance which normally stimulates them; they cease to grow, not because their power of growth is exhausted, but because of physical and chemical inhibitory influence. He believed that these inhibitory influences are chiefly chemical, and that in the case of the bones, for example, some substance accumulates in the body which checks the further growth of bone. This substance, he believed, is furnished by the hypophysis cerebri in minute quantities, probably from birth, and gradually accumulates until it is sufficient to counterbalance the inherent tendency of the bones, and perhaps of other tissues, to grow. When the pituitary body becomes diseased, and no more of this inhibitory substance is furnished, the bones, by virtue of their inherent tendency, become hyperplastic. Why this hyperplasia in acromegaly affects especially the peripheral parts of the skeleton and the face, he could not say, but this preference is not without analogy in other diseases.

The pituitary may have other functions beside those of inhibiting excessive growth of bony tissue.

Dr. Charles W. Burr believed that their case threw light on the subject of acromegaly. Cases of tumor of the pituitary body without acromegaly have been reported, and it has been believed from these gross examinations that disease of the pituitary body has nothing to do with acromegaly. This view is distinctly wrong. The sole object of their paper was to show that in order to prove that the pituitary body does not stand in a causal relation to acromegaly, it must be shown that in tumors of the pituitary body without acromegaly the substance of the organ has been completely destroyed by the new growth. When we recall that in fifty-seven out of sixty cases of acromegaly there was positive evidence of disease of the pituitary body, it must be admitted that there is some relation between the pituitary body and acromegaly.

Dr. Charles K. Mills reported a case of isolated paralysis of the ulnar nerve from unknown cause, and presented photographs.

CEREBRAL SYPHILIS AND SOME OF ITS MENTAL ASPECTS. A. E. Mink
(*Jour. of Am. Med. Assoc.*, 31, 1898, p. 5).

The author classifies the syndromes characteristic of brain syphilis as follows: 1. Syphilis of the base. 2. Syphilis of the vertex. 3. Syphilitic endarteritis which may be either basilar or vertical in situation. 4. Mixed forms, being both basilar and vertical in location and consisting usually of both specific endarteritis and meningitis. 5. Meningoencephalitis corticalis syphilitica. General symptoms common to nearly all varieties are: headache, especially in the earlier stages, and worse at night; insomnia and vertigo which may be complicated with nausea and vomiting. The patients are morose and irritable unless a syphilitic endarteritis predominates, in which case opposite conditions prevail, the stupor and drowsiness sometimes deepening into coma and even rendering the patient incapable of attending to the most pressing demands of nature. In many cases pronounced cardiac and gastro-intestinal irritability and a condition of vasomotor paresis of the cutaneous arterioles were observed.

The following phenomena are peculiar to both focal and diffuse forms of cerebral syphilis: 1. Sudden shifting of symptoms. 2. The predominance of cranial nerve palsies. 3. The multiplicity of palsies along with their coming and going. 4. The loss of particular accomplishments. 5. The concomitant appearance of symptoms due to destroying lesions of various cortical areas and the sudden coming or going of apparently fatal symptoms such as coma followed by perfectly lucid states. 7. The comparatively rapid termination in dementia. Mercury and the iodides are the sheet anchors in treatment, the former in the form of inunctions and the latter in some reliable syrup of hydriodic acid such as Gardner's, rather than the potassium or sodium salts, which, owing to the heavy dosage necessary, nearly always produce unpleasant symptoms, which may be entirely avoided by the use of the above preparation.

VOGEL.

Periscope.

ANATOMY AND PHYSIOLOGY.

- I. ON THE RELATION OF THE NERVOUS SYSTEM TO DISEASE AND DISORDER OF THE VISCERA. A. Morison (Edinburgh Medical Journal, vol. 3, 1898, pp. 124, 225, 374).

In these—the “Morison Lectures,” before the Royal College of Physicians of Edinburgh—the author discusses “The Anatomy and Physiology of the Nervous Mechanism of the Viscera,” stating that he expects to supplement them later by others devoted to the clinical aspects of the subject. The results of numerous authors are reviewed, and are criticized, in the light of his own experiences. In his histological work he has used the Golgi-Cajal method—as applied in Kölliker’s laboratory—and the method of Sihler, which, for tracing out the finest nerve fibers, he has found most useful. The stream of visceral innervation he likens to a delta with three mouths, these being respectively: 1, “The secreting cell;” 2, “the cell of involuntary muscle fiber,” and 3, “an intermediate or compound, and more obscure condition, the innervation of metabolism and excretion.” Just how the ultimate nerve fibrils end does not seem to be entirely settled, but the author inclines to the view that some at least end free in or among cells. It is probable that nerves to glands and muscles contain both efferent and afferent fibers, but it does not seem possible to distinguish one from another with any certainty.

Tracing back the fibrils to their plexuses in the viscera, the author discusses next the structure and function of the ganglia which occur in connection with these plexuses. He regards them in the main as stations for distributing, and perhaps for modifying, the centrifugal impulses. Proceeding still further toward the center, the structure of the sympathetic nervous system is taken up; its connection with central and peripheral nervous system is studied, and it is attempted to trace the passage of impulses through it. Lastly, the relations of the glosso-pharyngeus, vagus and accessorius are considered. The author then proceeds to discuss the physiology of visceral control, which, unfortunately, is not rendered very clear.

It is to be regretted that he could not find time and space in this series of lectures for the incorporation of his practical, clinical deductions.

The lectures are written in a scholarly manner, and are illustrated by numerous reproductions of photomicrographs. ALLEN.

2. THE OCCURRENCE OF NERVES ON INTRACRANIAL BLOOD VESSELS. G. L. Gulland (British Medical Journal, ii., 1898, p. 781).

The author has confirmed the assertion of Obersteiner (1897) that some of the vessels of the brain are supplied with nerves. He has found them on a number of vessels, and says they present the usual appearances of perivascular plexuses. He said further that Morison, of London, had been able to demonstrate them by Sihler’s hematoxylin method. In the discussion, Professor Stewart stated that, five months before, Huber, of the University of Michigan, had showed him preparations of blood vessels of the brain, stained with methylene-blue, and exhibiting nerve fibers in and around them.

PATRICK.

3. RECHERCHES SUR L'ORIGINE RÉELLE DES NERFS CRANIENS (Researches on the Real Origin of the Cranial Nerves). Van Gehuchten (*Journal de Neurologie*, 14, 15, 1898, pp. 273, 293).

According to Van Gehuchten, the facial nucleus in the rabbit consists of four divisions—the internal, middle, external, and dorsal groups, and these divisions exist almost throughout the entire length of the nucleus. In the uppermost portion of the nucleus the middle group disappears; in the lowermost portion the dorsal group is absent. In the dog the facial nucleus consists of only three groups, and these extend throughout its length. In the rabbit the peripheral neurons of the seventh nerve do not cross the raphe, and all the fibers of this nerve arise exclusively in the group of cells situated between the superior olive and the nucleus ambiguus.

Van Gehuchten found chromatolysis in the cells of the external, middle, and posterior groups, and of the external part of the internal group, after the seventh nerve in the rabbit was cut close to its exit from the stylomastoid foramen, before it had given off any extracranial branches. The cells of the internal part of the internal group, therefore, give origin to the fibers, which leave the seventh nerve before its exit at the stylomastoid foramen. From the results of similar experiments he concludes that the cells of the middle and external groups are connected with the fibers of the seventh nerve innervating the muscles of the face, except those contained within the upper branch of this nerve, and that the latter fibers arise in the posterior group. The ventral part of the seventh nucleus in the rabbit, therefore, represents the origin of the lower branch of the seventh nerve, while the dorsal part represents the origin of the upper branch.

The seventh nerve contains sensory fibers. If the central end of the cut facial nerve is irritated it causes pain. The chromatolysis which Van Gehuchten found in a few cells of the geniculate ganglion, after the seventh nerve had been cut at its exit from the stylomastoid foramen, shows that the geniculate ganglion is a cerebrospinal ganglion, and that it gives origin to the sensory fibers in the seventh nerve at its exit from the Fallopiian canal. The nerve of Wrisberg is the sensory root of the seventh nerve. Amabilino obtained results in the dog different from these obtained by Van Gehuchten in the rabbit. The former believes that the cells of the geniculate ganglion send their peripheral processes into the chorda tympani nerve, and that none of these pass into the extracranial branches of the seventh nerve

SPILLER.

4. CONTRIBUTION A L'ÉTUDE DES LOCALISATIONS DES NOYAUX MOTEURS DANS LA MOELLE LOMBO-SACRÉE ET DE LA VACUOLISATION DES CELLULES NERVEUSES (Contribution to the Study of the Localization of the Motor Nuclei in the Lumbo-sacral Cord and of Vacuolization of Nerve Cells. A. Van Gehuchten et de Buck (*Revue Neurologique*, 6, 1898, p. 510)

The authors discuss the question of the nuclei of origin of the motor nerves of the muscles of the leg and of the foot, coming to the following conclusions:

(1.) The nuclei of origin of the motor nerves of the muscles of the leg and foot occupy the posterior part of the anterior cornua of the cord and extend from the upper portion of the fifth lumbar segment to the lower portion of the fourth sacral segment.

(2.) Two large nuclei are found which serve to innervate these portions. The first, very large, probably made up of several divisions,

extends from the upper border of the fifth lumbar segment to the lower part of the third sacral segment; a second nucleus equally large, especially at its center, commences, behind the first, at the upper level of the second sacral segment and extends to the lower extremity of the fourth sacral segment.

Under the caption of vacuolization, the authors consider the cause of this very common occurrence. It is their opinion that such vacuolization is not an inherent lesion of secondary degenerations, as taught by Marinesco, nor is the post-mortem theory with its many adherents altogether tenable; the artefact hypothesis does not meet with the authors' approbation, and they leave it in much the same condition that it is, stating that in the actual state of our knowledge a definite answer as to the cause of this appearance is impossible.

JELLIFFE.

5. UEBER DIE VERÄNDERUNGEN DER MOTORISCHEN FUNCTIONEN BEI STÖRUNGEN DER SENSIBILITÄT (Concerning Disturbances of Motion Following Disturbances of Sensation). A. v. Korniloff (Deutsche Zeitschrift für Nervenheilkunde, 12, 3 and 4).

In the dog the sensory and motor nerves of the posterior limbs are represented in the fourth, fifth, sixth and seventh lumbar and first sacral roots; occasionally also in the third lumbar and second sacral. It is necessary, therefore, to cut five roots to cause anesthesia in a posterior limb of a dog, and it is safer to cut all seven. Korniloff believes, from his experiments on a number of dogs, that division of the posterior roots causes disturbance of motion corresponding in intensity to the number of roots cut, and that when all the posterior roots of a limb are cut the limb is almost completely paralyzed. When some, but not all, of the sensory roots of a limb are cut, the movements are ataxic. The disturbance of motion is dependent on the degree of the disturbance of sensation. The writer believes that the clinical phenomena observed in these experiments have some bearing on the symptomatology of tabes.

SPILLER.

PATHOLOGY.

6. CONTRIBUTION A L'ÉTUDE DES DEGENERESCENCES PROPAGÉES ET EN PARTICULIER DES ALTÉRATIONS DES CORDONS POSTÉRIEURS CONSECUTIVES AUX LÉSIONS EN FOYER DE L'ENCÉPHALE (Contribution to the Study of Extending Degenerations, in Particular, Alterations in Posterior Columns Following Lesions in the Brain). G. Durante (Revue Neurologique, 6, 1898, p. 390).

From a study of four cases, three being of hemiplegia, the author brings out, what is becoming more and more evident, that retrograde degenerations do not always stop at the "relay" ganglia; that they are not necessarily confined to a single neuron, and that centrifugal sensory degeneration and centripetal motor degeneration may occur. He particularly calls attention to the occurrence of retrograde degeneration of the columns of Goll and Burdach following cerebral lesions, of which he has collected some twenty-three cases reported in literature and interpreted in a number of ways by their authors. Of twenty-nine cases, six of the author's, seventeen such types of degeneration were noted in lesions of the pons, the cerebral peduncles or the central gray nuclei; twelve were consecutive to lesions of the cortex.

From the clinical side, these observations are of interest from the relationship of such degenerations to cases of high tabes and to a number of irregular cases generally designated tabetiform.

JELLIFFE.

7. LESIONS DES CELLULES DE L'ECORCE CEREBRALE DANS CERTAINES FORMES DE CONFUSION MENTALE (Lesions of the Cells of the Cerebral Cortex in Certain Forms of Mental Confusion). Gilbert Ballet (La Médecine Moderne, 9, 1898, p. 421.)

Under the name of mental confusion, a complex of symptoms is described which, until lately, has been confounded according to the most marked symptoms which each case might present, with febrile deliriums, sometimes with the deliriums of degeneration, sometimes with the different forms of melancholia, particularly with the stuporous variety. The predominating symptom is a confusion of ideas. Speech, when the patient talks, is loose, confused, sometimes almost incomprehensible. There is a manifest difficulty in the association of ideas, and an evident obstacle to intellectual function. The patient wants decision, memory is bad, power of attention impaired, the notions of space, place and time are enfeebled or wanting. There are usually hallucinations, and the patient lives in a kind of half-automatic activity; even if there are sane ideas, they are vague and changing, without system or constancy. This condition of things frequently follows infectious or toxic diseases.

All these symptoms point to its dependence upon lesions of the cortex, but up to the present no decided cortical lesions have been observed, or, at least, none more marked than thickening of the pia or atrophy of some myelin fibers.

Ballet has studied two cases presenting almost identical symptomatology. Both were women, both alcoholics, and both had slight tubercular lesions of the apices of the lungs; one presented some signs of polyneuritis of the legs, and both presented the complete mental picture of the disease under discussion. The post-mortem examinations of the brains were almost identical in their results. The cortex, stained by Nissl's method, was observed with a low power, and the large pyramidal cells, especially the cells of Betz, appeared to have lost their triangular form and to have become rounded. The processes were less marked, and even with a low power it was possible to observe the displacement of the nucleus, while with a high power it was only here and there a sound cell could be found. Chromatolysis was going on in all of them, the nucleus seemed to be pressing out of the cell, and in some of them the chromatolysis was complete and the nucleus entirely lost to view. It is possible that these changes were secondary and consecutive to the destruction by the poison of the cylinder axes. The analogy which they present to the lesions consequent upon experimental section or other pathological changes of the peripheral nerves, and the absence of vascular alteration, make this hypothesis probable. Meanwhile, it is interesting to find decided cellular cortical lesions in a disease hitherto classed as a psychosis.

MITCHELL.

8. BEITRAG ZUR PATHOLOGISCHEN ANATOMIE DES CENTRALNERVENSYSTEMS BEI DER ACUTEN ANÄMIE (Contribution to the Pathological Anatomy of the Central Nervous System in Acute Anemia). G. Scagliosi (Deutsche med. Wochenschrift, 20, 1898, p. 309).

Until the present time, no satisfactory report has been made concerning the condition of the ganglion cells in acute anemia, such as is produced by severe hemorrhage. Scagliosi reports the results of an examination of the brain and cord from a pregnant woman, who had uterine hemorrhage lasting thirty-four days. The gray matter was pale and the vessels were not as full as under normal conditions. The

cells of the brain and cord presented chromatolysis. He believes that his case shows that a diminution in the number of red blood corpuscles is sufficient to cause intense alteration of the ganglion cells.

SPILLER.

9. SULLE ALTERAZIONE ISTOLOGICHE DEL SISTEMA NERVOSA CENTRALE DEL FETO IN SEGUITO AD AVELLENAMENTO ACUTO E CRONICO DELLA MADRE PER ALCOOL (Histological Alterations in the Nervous System of the Fetus, Following Acute and Chronic Alcohol Poisoning in the Mother). M. Carrara (Rivista di Medicina legale e di Giurisprudenza med., vol. 6, 1898, No. 2).

In pregnant guinea pigs poisoned by alcohol, the author shows that in the central nervous system of the young pigs after fixation by corrosive sublimate and staining by the methods of Nissl, Bizzozero, and Heidenhain that there are certain cell changes which, as far as the investigation has proceeded, have been constant.

In the brain the changes noted were but slight; they consisted for the most part of a lack of colorability of the chromatic substances. The nuclear membrane was also less distinct. Many karyokinetic figures were shown by the Bizzozero methods of staining. In the anterior horn cells of the spinal cord the changes were more pronounced. In them there was a marked peripheral chromatolysis similar to that found by Marinesco and others in direct poisoning of the animal itself.

JELLIFFE.

10. UEBER MORPHOLOGISCHE DIFFERENZEN ZWISCHEN RUHENDEN UND ERREGTEN GANGLIENZELLEN (Concerning Morphological Differences Between Resting and Irritated Ganglion Cells). Friedel Pick (Deutsche med. Wochenenschrift, 22, 1898, p. 341).

Direct electrical irritation of the peripheral neurons must produce results differing from those occurring after normal action. Pick, to imitate normal conditions as closely as possible, exposed the brain in monkeys and cats when they were in narcosis and irritated it with the faradic current for a half hour to one hour, so that contractions occurred only on one side of the body. During the irritation the spinal canal was opened, and the segment necessary for the production of the movements was determined approximately by successive division of the roots or spinal cord. Sections from this and other portions were stained by the methods of Nissl and Golgi. The irritation in this way was applied at a remote point from the spinal cells. As only the cells of one side of the cord were irritated, a contrast was offered in the same preparation between the active and the resting cells. The former presented chromatolysis, chiefly peripheral, and this Pick believed favored the view that the chromatic substance is reserve material and disappears during activity. The cells were of irregular contour, and contained shrunken nuclei. In preparations from monkeys the cells near the union of the anterior and posterior horns were most altered, and this fact Pick believes proves v. Monakow's theory, according to which the central and peripheral motor neurons are connected by short interposed neurons.

SPILLER.

CLINICAL NEUROLOGY.

11. LES ACCIDENTS NERVEUX DU DIABETE (The Nervous Accidents of Diabetes). J. Vergely (Archives Cliniques de Bordeaux, Vol. 7, 1898, Nos. 3, 4, 5 and 6).

In this article of 154 pages, the author reviews very thoroughly

the nervous complications of diabetes, taking up the subject under the following heads:

- I. Troubles of motility.
- II. Troubles of sensibility.
- III. Troubles affecting the genital organs and organs of the senses.
- IV. Vasomotor and trophic disturbances.
- V. Cerebral troubles.
- VI. The different varieties of diabetes, from a nervous point of view.
- VII. The rôle of the heart in the production of nervous accidents.
- VIII. Diabetic coma.
- IX. The lesions of the nervous system in diabetes.
- X. The relation of diabetes to neuroses and other nervous diseases.

It is clearly shown that a large number of the symptoms of nervous disease may occur in connection with diabetes, some of them from causes acting temporarily (probably toxemia); others from organic lesions of the nervous system, dependent to a greater or less extent upon the constitutional disease present. In the brain, hyperemia, hemorrhage and embolic softening have been observed; involvement of the spinal cord is rare, while of all changes in the nervous system neuritis is most commonly found.

This last will probably explain many of the nervous symptoms. The author emphasizes the necessity for careful urinary examination in very obstinate neuralgias, and in a typical paralysis, sensory and trophic disturbances.

Of special interest is the account of "diabetic pseudo-tabes," the cerebral disturbances occurring in diabetics, and the various theories with regard to diabetic coma. To all specially interested a study of the original article is recommended, as it contains a very thorough résumé of the subject, with a complete bibliographic index. ALLEN.

12. A CASE OF ACROMEGALY. Kauffman (Birmingham Medical Review), vol. 43, 1898, June.

A man of twenty-three years, showing the ordinary enlargement of the hands and face, the feet being normal. The fields of vision were normal. The beginning of the disease dated back a twelve-month, headache being the first symptom, followed by overgrowth of face and hands and antero-posterior curvature of the spine.

PATRICK.

13. THE CRANIUM OF THE INSANE. (Osteitis Deformans and Acromegaly). Beadles (Edinburgh Medical Journal, vol. 3, 1898, Nos. 3, 4, 5).

The author, from a study of asylum records and from 234 autopsies performed by himself upon insane patients, concludes that in chronic insanity thickening and increase in weight of the cranial vault is exceedingly frequent, especially in those types characterized by periodical excitement.

The nature of the process causing this thickening is studied and compared to that in osteitis deformans. He then describes the condition of the skull in three cases of this disease—one from his own notes—and in two cases of acromegaly, comparing them and calling attention to the difference in character and location of the hypertrophic process in the two diseases. The article is illustrated by two plates and several wood cuts. ALLEN.

THERAPY.

14. GOITRE EXOPHTHALMIQUE TRAITÉ PAR LA RÉSECTION DES DEUX SYMPATHIQUES CERVICAUX (The Surgical Treatment of Exophthalmic Goitre, Bilateral Resection of the Cervical Sympathetic). M. Gérard-Marchant et Ch. Abadie, also Chauffard et Quénu, *La Presse Médicale*, vol. 5 (2), 1897, p. 1, July 3; M. Jonnesco, *ibid*, vol. 5, Oct. 23, 1897, p. 257.

Proceeding upon the theory that the symptoms of exophthalmic goitre are due to the excitement of the vaso-dilators through the cervical sympathetic, the first mentioned authors practised bilateral resection of this nerve in the case of a woman of twenty-nine, in whom the disease had been present for about a year. This patient had marked exophthalmos, moderately enlarged thyroid, tremor, loss of flesh, pulse about 100. An incision was made along the posterior border of the sterno-mastoid muscle, from the mastoid process to the clavicle; the sympathetic was sought out, and part of the superior cervical ganglion, with three and a half to four centimeters of the portion of the nerve peripheral to it, was removed, first on one side, then on the other. The pupils were not altered at the moment of cutting the nerve. Just after the operation the pulse was 80. Five days later the exophthalmos had nearly disappeared, and the patient was much improved. The further course of the case is not stated.

The case of Chauffard and Quénu was that of a man of twenty-four, in whom the first symptoms of the disease were noticed three years before he came under their observation. He had marked exophthalmos, moderate sized goitre, pulse 110, and other symptoms of Basedow's disease. As he did not improve under general treatment, both cervical sympathetics were resected under the same technique as that practised in the case of Gerard-Marchant and Abadie. The section was followed by no vasomotor phenomena, but the pupils narrowed immediately. The case was kept under observation for about two months after the operation, but the authors were unable to convince themselves that any improvement had occurred. The patient died soon after from an overdose of digitalin which he had carelessly taken.

Jonnesco gives a résumé of the surgical treatment of exophthalmic goitre, dividing the operations practised into: a, those upon the thyroid gland; b, those upon the cervical sympathetic, and c, ligature of the thyroid arteries. His conclusions are: 1. Operations upon the thyroid gland, while benign in simple goitre, are very dangerous in exophthalmic goitre. They can accomplish no good except in old goitres which have given rise to symptoms secondarily, but which do not in strictness constitute Basedow's disease. 2. Simple section of the sympathetic is useless. Partial resection, if including the superior and middle cervical ganglia, offers better results. 3. Total bilateral resection is the operation of choice. Its ease and benignity are such that it should always be tried in cases of exophthalmic goitre, while partial thyroidectomy remains an operation of necessity, to be resorted to only in the presence of grave symptoms which demand a rapid removal of the tumor. He reports three cases, all in women. In two of them he made a partial, in the third, a complete bilateral resection, including all three cervical ganglia of the sympathetic. The two former cases he has been able to observe for fourteen months; the latter case for three months. In all, the frequency of the pulse is reduced, the exophthalmos and goitre, the tremor and other nervous symptoms have disappeared, and he thinks a cure can justly be claimed. ALLEN.

15. A CASE OF EXOPHTHALMIC GOITRE WITH UNILATERAL EYE SYMPTOMS. J. Hinshelwood (British Medical Journal, 1, 1898, p. 1653).

A single woman of twenty-four years presented left exophthalmos and some enlargement of the thyroid gland without tachycardia. Stellwag's symptom (retraction of the upper lid) and Graefe's sign were present, and the patient had become somewhat nervous. Upon five-grain doses of antipyrin t.i.d. there was evident improvement after three weeks, and in five weeks the exophthalmos and Stellwag's sign had disappeared, the thyroid gland had markedly diminished in size, but, what is noteworthy, Graefe's sign persisted. The dose of antipyrin was gradually increased to thrice the initial amount.

PATRICK.

16. ON DIFFUSE SCLERODERMA, WITH SPECIAL REFERENCE TO DIAGNOSIS AND TO THE USE OF THE THYROID GLAND EXTRACT. W. Osler (Journal of Cutaneous and Genito-Urinary Diseases, 16, 1898, Nos. 2 and 3).

In this communication, based on the study of eight cases seen by him, Professor Osler considers the subject under the following heads: 1. General picture of diffuse scleroderma. 2. Scleroderma and Graves' disease. 3. Differential diagnosis. 4. Scleroderma and Addison's disease. 5. The treatment of scleroderma with thyroid extract. His description of the disease, its diagnosis and relationships, as illustrated by the cases described, is interesting and instructive.

Six of the eight cases received thyroid treatment. The author concludes that his personal experience, and the results as recorded in literature, do not favor the use of thyroid gland extract. ALLEN.

17. MÉDICATION THYROÏDIENNE ET ARSENIC (Thyroid Medication and Arsenic). MM. Bedart et Mabilie (La Médecine Moderne, vol. 9, 1898, p. 335).

MM. Bedart and Mabilie made an experimental study of the combined use of thyroid preparations with arsenic, and found that the use of arsenic seemed to perfectly control the excitement, the palpitation, and the tremor which are so troublesome when thyroid alone is given. Their experiments upon animals included fourteen dogs and two rabbits, and they sum up their results as follows: The animals which did not receive any arsenic had a rapid increase of the speed of the heart, going from 130 to 180 and 190 with very irregular action. The animals taking in addition to thyroid Fowler's solution in a dose of 2½ to a kilo. of the animal's weight had at first a stationary condition of the pulse, and then a decrease in the number of beats from 130 to 100, and sometimes even to 90, with the contractions remaining normal in force and regular. After six or seven days of thyroid feeding, the animals not receiving arsenic presented phenomena of excitability and of generalized muscular tremor, but none of these troubles were observed in those that received arsenic as well, even after thirty days of the experiment. A considerable loss of weight manifested itself very rapidly in the dogs which were taking nothing but thyroid without arsenic. Those receiving arsenic lost also, but more slowly. Some of them did not lose at all, and one or two even gained a little.

It is to be noted that the dose of arsenic was one which would be regarded in medical practice as an enormous one. MITCHELL.

18. EXOPHTHALMIC GOITRE TREATED WITH THYMUS GLAND. P. James (Australasian Medical Gazette, vol. 16, 1897, p. 337).

The author reports the following case: A woman of twenty-eight presented the classical symptoms of Graves' disease and had lost much flesh and strength. She was treated with a 50 per cent. glycerine extract of thymus gland, in doses of a half teaspoonful to a teaspoonful, three times a day, for about a month. She experienced at times a sensation of depression shortly after taking the extract, and both before and after the treatment was begun she had from time to time attacks of vomiting and diarrhea, which prostrated her very much. Later tabloids of thymus gland were substituted for the extract. The initial dose was two tabloids three times a day. This was gradually increased until it was found that five tabloids three times a day was the maximum safe dose. She continued to take the tabloids for nine months. The tremor, sweating and pulsation gradually grew less, the pulse was slower, the patient gained weight and strength, and Graefe's and Stellwag's signs disappeared. Ten months after the treatment was begun she was able to eat heartily and to sleep well, and could undergo a fair amount of fatigue. Her pulse ranged from 84 to 88. The exophthalmos and goitre still persisted, but to a less degree. The other symptoms had disappeared.

ALLEN.

19. THE THERAPEUTIC VALUE OF SPLEEN EXTRACT. C. Clark (Edinburgh Med. Journal, 3, 1898, p. 152).

The frequency with which smallness of the spleen is noted in the post-mortem records of insane asylums led the author—the superintendent of the Lanark County Asylum—to try the effect of spleen extract in the treatment of insanity. A fluid extract made from fresh bullock's spleen, an emulsion of the ethereal extract, and compressed tablets were employed.

About thirty cases were subjected to the treatment. Concerning these he makes the general statement that some were cured, others benefited, still others unaffected, but of only six does he give a detailed account.

Of these latter, five were more or less improved, one cured, but he is not at all sure that the result is to be attributed to the spleen extract. Under the use of the remedy, he noticed a general improvement in appetite and digestion, more or less gain in weight, improved blood condition, and specially improvement in nutrition of the skin, cold, dry and harsh skins becoming warm, moist, soft, elastic and of better color. On account of this latter fact he was led to try the extract in some cases of chronic skin diseases, with satisfactory results. In general, the extract seemed to have some stimulant effect, and, what is noteworthy, markedly increased the susceptibility of the patient to thyroid extract. Hence the author advises a preliminary course of splenic extract in cases which are later to receive thyroid, the latter being then given in much reduced doses. He has observed no toxic symptoms, but advises care, especially in the use of the ethereal extract, which he regards as the most active preparation.

ALLEN.

- A DISCUSSION OF THE TREATMENT OF INTRACRANIAL TUMORS. David Ferrier. (British Medical Journal, 1898, No. 1970, p. 964.)

The percentage of operable tumors has been variously estimated to be from two (Seydel) to fifteen (Beck) per cent. The speaker considers seven per cent. a fair estimate.

Regarding the results of operation he first submitted statistics

of fifty-five cases operated on since January 1896, and published in various journals. Of these, twenty (sixteen cerebral and four cerebellar) died within a few hours after the operation, sixteen (eleven cerebral and five cerebellar) died within a year, twelve (ten cerebral and two cerebellar) recovered from the operation and passed from the hands of the surgeon, seven (six cerebral and one cerebellar) were alive a year or more after the operation. The last are reckoned as cured and constitute thirteen per cent. of the total.

The statistics compiled from the records of the National Hospital for the Paralyzed and Epileptic were more valuable. Total number of cases operated upon in the last ten years, thirty-eight (thirty-one cerebral and seven cerebellar). In twenty of these (sixteen cerebral and four cerebellar), the growth was removed wholly or in part, while in eighteen (fifteen cerebral and three cerebellar), it was not removed. Of those removed, three died within a few days, seven died within a few weeks or months, four lived some weeks or months, the subsequent history being unknown, and six (i. e., sixteen per cent. of all operated, thirty per cent. of those in which tumor was removed) lived a year or more, the longest period being four years. Of the eighteen cases in which the growth was merely exposed or exposed and incised, three (16.6 per cent.) lived a year or more, one of these, a cerebellar case, being alive after six and a half years.

A patient whose cerebellum was incised, but whose tumor was not found, wrote after six years:

"I am pleased to tell you that with care my health is quite satisfactory. I still have the swelling at the side of my head, but it causes no inconvenience except being rather sensitive to touch. I also still have the weakness down the right side, but this is much better than before I left the hospital. Indeed, it will give you an idea how much better I am when I tell you I have been able to cycle for two seasons."

The speaker agreed with Horsley that gummata and gummatous cicatrices which will not yield to vigorous antispecific treatment should be operated.

F. X. Dercum believed with Ferrier in the efficacy of potassium iodide in glioma of the brain and in the utility of trephining and simply opening the dura in inoperable cases.

Joseph Collins suggested that lumbar puncture be employed early and often in the hope of decreasing intraventricular pressure.

John H. MacCormac commended removal of gummata by the knife when specific treatment failed and said that when it was remembered how extremely tough such a tumor might be, it was not wonderful that iodides failed to cause its absorption.

Headache might be much relieved by phenacetin gradually and carefully given in doses up to gr. xx combined with caffeine gr. v, repeated if necessary in three hours. Vomiting might be relieved by giving $\frac{1}{16}$ gr. of hydrobromate of hyosine every four hours. Mercurial treatment might be tried not only in cases of syphilitic tumor, but also for glioma and sarcoma.

C. E. Beever had in four cases seen the tumor removed and the patient live two years or more.

Herbert E. Waterhouse cited a case showing that a gumma may not be amenable to specific treatment. After administration of potassium iodide and mercury for four weeks, the patient was put on twenty-five grains of iodide t. i. d., but in spite of this the symptoms increased until he was comatose, when a large gumma was removed from the Rolandic area by operation. Recovery was rapid and complete. The speaker also reported palliation in several cases of operation in which the tumor could not be removed or was not found.

J. Mitchell Clarke and J. M. Cotterill also commended the palliative operation.

Byron Bramwell said that his experience pointed very strongly to the conclusion that it was only in a very small proportion of cases that an intracranial tumor could be completely and satisfactorily removed. An analysis of all the cases seen by him in twenty-five years brought out the following facts:

In 2 cases a tumor was found, the presence of which was not suspected during life; in other words, in which there were absolutely no symptoms. In 23 cases there were well-marked "general" symptoms, but no localizing symptoms. In 2 cases, in which very definite localizing symptoms were present, no tumor was found at the operation. In one of these the symptoms clearly pointed to a tumor in the motor area; at the post-mortem examination, a tumor was found in the optic thalamus, pressing upon and involving the motor strands of the internal capsule. In another case, in which the symptoms clearly seemed to point to the cerebellum as the seat of the lesion, no tumor was found, although the patient was trephined first over the left and then over the right lobe of the cerebellum. In 25 cases the position of the tumor precluded successful operative procedure; in 3 of these cases the tumor was situated at the base of the brain, outside the pons Varolii; in 4 the tumor involved the pituitary body; in 12 the pons Varolii, and in 6 the crus cerebri. In 9 cases, not included under any of the other headings, the cerebellum was the seat of the lesion; but it was very doubtful whether in all or any of these cases the tumor could have been successfully removed. He would refer to this point again presently. In 11 cases the tumor, as shown by the symptoms during life, by actual operation, or on post-mortem examination, was found to be too extensive to permit of removal. In 3 cases there were multiple growths. In 8 cases the tumor was malignant—cancer or melanotic sarcoma. In 1 case in which a healed syphilitic gumma was the cause of sensory Jacksonian epilepsy, the irritating cause might have been removed by operation, but malignant disease (epithelioma of the tongue) was present, and precluded operative procedure. In 31 cases the symptoms were relieved by medicinal treatment, and an operation consequently rendered unnecessary. In 24 of these cases the tumor was syphilitic; in the remaining 7 syphilis could be definitely excluded. In one of these 7 cases the surgeon who was consulted refused to operate; the patient improved markedly for a time under treatment (iodide of potassium), but relapsed and died soon after his discharge from hospital. In 5 of the remaining cases in which the symptoms had entirely, or almost entirely, subsided up to the present time, the exact nature of the pathological condition was perhaps doubtful. Dr. Bramwell used to be in the habit of regarding cases of this sort as cases in which the tumor had become quiescent; but latterly he had come to doubt whether this opinion was correct, at all events in all cases of the kind; he was now disposed to think that in some of them, at all events, the pathological lesion was probably a dilated condition of the ventricles, due to localized meningitis and closure of the foramen of Majendie. He would refer to this point again presently. In 1 case in which the symptoms pointed very definitely to a localized tumor in the motor area no tumor was found at the operation, though post-mortem examination subsequently showed a small glioma in the suspected situation; it was situated about $1\frac{1}{2}$ inches below the surface. In the remaining 6 cases an operation should perhaps have been performed. In one of these cases (a scrofulous tumor of the cerebellum)

the patient refused to allow of operation. In 2 cases the patient was only seen once and it was not known whether the symptoms were relieved by drug treatment or not. In the remaining 2 cases in which there was no post-mortem examination, the tumor appeared to be situated in the frontal lobe; in 1 of these cases the tumor seemed to be an extensive infiltrating glioma. In another case the tumor was situated in the occipital lobe; this patient was only seen once in consultation and there was no post-mortem examination.

Dr. Bramwell's pathological experience entirely corroborated his clinical observations and showed that very few cases of intracranial tumor which were examined post mortem could have been successfully removed by the surgeon. There were of course fallacies, for at the date of the necropsy the tumor was often very much more extensive than in the early (clinical) stages of the case. Further, tumors which one observer considered unsuitable for removal might by another observer be considered suitable.

Dr. Bramwell thought the question of operation was especially difficult in cases of cerebellar disease. He had met with at least 3 cases in which all the characteristic symptoms of a cerebellar tumor were present; but in which post-mortem examination showed, instead of the tumor which had been diagnosed, that the lesion was a dilated condition of the ventricles, apparently due to a localized meningitis which had blocked the foramen of Majendie. In one of these cases the patient was trephined. In all of these 3 cases the symptoms were of considerable duration (several months); in one of them the symptoms followed an attack of influenza, and in that case there was febrile disturbance during the early stages of the case; in the other 2 cases there was no history of fever and no symptoms indicative of meningitis. These cases seemed to suggest that in some of the cases at all events in which a cerebellar tumor had been diagnosed, and in which the symptoms subsided and the patient got well, the diagnosis was perhaps erroneous. He thought it probable that in some of these cases the pathological lesion was probably a dilated condition of the ventricles.

His experience entirely coincided with that of Dr. Trevelyan—namely, that it was exceedingly rare to meet with healed tumors, or cases in which the tumor had become encapsuled, latent, and quiescent, in the post-mortem room. During the whole course of his pathological experience he had met with only 1 case in which there was reason to suppose that a healed tumor was present. He did not, of course, in this statement include the syphilitic cases.

Now, if, as his experience clearly showed, cases were not very uncommon in which patients who presented all the general symptoms of a cerebellar tumor got well, it must, he thought, be granted that if in these cases the diagnosis was correct the remains of these healed tumors ought to be much more frequently found after death than appeared to be the case. Consequently, as the result both of his clinical and pathological experience he concluded that in some of these cases, at all events, the pathological lesion was a localized meningitis with ventricular distension and increased intracranial pressure and not a cerebellar tumor. The question was one of great practical importance, for if this conclusion were correct one would necessarily hesitate to recommend operative procedure in cases of supposed cerebellar tumor until drug treatment had been very fully tried for a considerable length of time. But if this course were adopted, one ran some risk of the patient dying suddenly, as some of the cases to which Professor Ferrier referred had done, and as had happened in more than one case which had come under his own observation. Hence one was on the horns of a dilemma.

Further, as Professor Ferrier had pointed out, the dangers of operating on cerebellar tumors appeared to be considerably greater than in tumors of the cerebral hemispheres. Again, in some cases of cerebellar tumor, the middle lobe was involved, or a tumor of the lateral lobe was so extensive that its complete removal was very difficult or impossible. Again, in many cases of cerebellar disease it was extremely difficult or impossible to determine during life the exact part (lobe) of the cerebellum which was involved; and a double operation, first on one lateral lobe and then on the opposite lateral lobe, was undoubtedly attended, so far as his experience enabled him to judge, with considerable risk to life.

For all these reasons, although undoubtedly in some cases of cerebellar tumor the tumor might be removed, and brilliant results obtained by operation, the proportion of such cases seemed to him to be exceedingly small.

Four years ago he had published a list of 82 cases of tumor of the brain, and analyzed them for the purposes of a similar discussion. He had concluded that in only 5 of these would an operation have, as far as he could judge, been attended with success, even if it had seemed to be advisable (and in most of these cases this did not seem to be the case) while the patient was under his observation.

Since these statistics were published he had met with 41 additional cases, and in none of them, although he had been on the look-out for cases suitable for operative treatment, had the tumor been successfully removed by surgical procedure. As a matter of fact, an operation was performed in 14 of his cases. In some of these 14 cases the object aimed at was the removal of the tumor; in others, in which there was no definite localizing symptoms, the operation was performed merely as a palliative measure (for the relief of headache, etc.); and in others (although the symptoms seemed to preclude successful removal of the tumor) on the off-chance that the diagnosis of tumor might be erroneous, and that an abscess or some other removable lesion would be found.

Dr. Bramwell's experience, therefore, in regard to the success of operative procedure differed notably from that of Professor Ferrier, for in none of his fourteen cases in which an operation had been performed had a tumor been successfully removed by the surgeon. He could only account for this difference by supposing that he had had an unfortunate run of cases (a series of cases unsuitable for operation), or that Professors Ferrier and Victor Horsley had made a selection of their cases, rejecting those (such as infiltrating glioma) which their wide knowledge and experience seemed to show were unsuitable for operation. It was only in one or other of these ways, or by supposing that their experience enabled them to localize cerebral tumors more accurately, or to remove new growths more satisfactorily, than himself and some of the other observers who had published their results, that one could account for the difference in the results to which Dr. Collins had referred.

Though in some cases marked benefit had been obtained by trephining for the relief of symptoms, he had, on the whole, been somewhat disappointed with the result of those cases in which "palliative" operations had been performed. He entirely agreed, however, with Professor Ferrier and the other speakers in thinking that if, after a reasonable time, drug treatment failed to give relief, an operation should be performed, even although the conditions for operation did not appear to be satisfactory or hopeful. In the vast majority of cases of intracranial tumor (the syphilitic cases, of course, excepted) the only chance, of course, was operative procedure.

PATRICK.

Book Reviews.

UNTERSUCHUNGEN ÜBER DEN LEPRABACILLUS UND ÜBER DIE HISTOLOGIE DER LEPRO. Victor Babes, Professor of Pathological Anatomy and Bacteriology at the University of Bucarest. S. Karger, Berlin, 1898, pp. 112.

The previous work of the author on the subject of leprosy makes this later monograph particularly welcome. Babes has here collected and arranged in systematic form his own work and that of others in the same field, and in such a way that a maximum ease of reference has been attained so far as that is possible without an index. After a discussion of the histology of leprosy, before the discovery of the causative agent, he takes up in detail the now recognized bacillus of the disease, its method of entrance into the tissues and its position in them. Then follows a consideration of leprosy as it appears in the skin and upper air passages, in the nervous system, genital organs, lymph and blood making organs, lungs, digestive tract and other tissues of less importance. The final chapter contains an excellent summary of conclusions, which is followed by a bibliography.

Of special interest to the neurologist is the discussion of changes in the nervous system, due to the presence of the bacillus of leprosy. Pathological alterations are frequent and easy of detection in the peripheral nerves, and are essentially what one would expect from the general character of the leprosy process. The bacilli are to be seen in and about the nerve trunks affected. Babes has been able to verify the researches of Sudakewitsch as to the presence of bacilli within the cells of the spinal ganglia, and also, though much less frequently, in the ganglia of the sympathetic system. As to the changes brought about in the cells by the presence within them of bacilli, Babes is not inclined to be dogmatic, though in general he found that those cells in which most marked alterations were observed contained bacilli. It is easy to conceive that, apart from the mere mechanical effect, the presence of organisms within the cell might produce no marked chromatolysis.

Observations on the spinal cord have been somewhat less satisfactory, and the cases examined more open to doubt in their interpretation. Contrary to previous researches by Chasiotis, Babes finds few bacilli in the cord, and when found they lie within nerve cells. In certain cases both the chromatic substance and the nucleus show evidence of degenerative changes. Researches on the brain have been, for the most part, negative.

Babes is entirely sceptical, and rightly we think, as to the relationship between leprosy and syringomyelia. His own words in the matter are: "We must in this important question make the absolute demand that preparations be shown us in which we may convince ourselves that bacilli are actually in the spinal cord, and then that they stand in causal relation to the cavity formation."

In general, the monograph is an important addition to our knowledge of a subject of the highest scientific interest, and may well find a

place in the library of the neurologist, as well as of the syphilographer. It is well printed, and profusely illustrated with colored plates, admirably reproduced from the originals. The proof-reading leaves something to be wished for, and the absence of an index is a real defect.

E. W. TAYLOR.

ACCIDENT AND INJURY; THEIR RELATIONS TO DISEASES OF THE NERVOUS SYSTEM. By Pearce Bailey, A.M., M.D. D. Appleton & Co., New York, pp. 12, 430, 1898.

There should be two *raisons d'être* for every medical book. First, the author should be so full of his subject by reason of large experience, careful review of the literature and mature deliberations as to make his writings the natural expression of accumulated wisdom; and second, there should be an expressed want or a real need on the part of the medical public for such a book. In the present instance it may well be doubted if the first *vis a tergo* was present in all its completeness; there can be no manner of doubt as to the second. A good monograph regarding the "traumatic neuroses" was sadly needed, and this need Dr. Bailey has supplied, notwithstanding the insinuation just made that the work bears some marks of immaturity. It is, indeed, something more than a treatise on that group of functional nervous disorders called by Oppenheim traumatic neuroses.

After an introduction devoted to general considerations regarding the principles and technique involved in a consideration of traumatic nervous cases the work is divided into four parts, as follows: (1) Organic Effects of Injury to the Nervous System (140 pages); (2) Functional Effects of Injury (155 pages); (3) Malingering (52 pages); (4) Treatment of the Traumatic Neuroses (24 pages).

It will be seen by this division of the subject that organic affections receive considerable attention, and this is as it should be, for in very many injury cases the first and sometimes the most difficult question to be resolved is, whether the patient is suffering from organic or functional disease, or both. To make this accurate and complete diagnosis a knowledge of both classes of affections is clearly indispensable.

In the introduction will be found detailed instructions as to what and how to examine in a case of injury when the nervous system is involved.

The author divides the examination into four stages, viz.: (1) Previous history of the patient (ancestral; personal); (2) history of the accident; (3) physical evidences of predisposition to nervous disease; (4) examination for the actual injury.

To this, in our opinion, should be added a separate head embracing the history from the accident to the time of the examination, as this part of the anamnesis is not infrequently the most important and often reveals the essential nature and pathogenesis of the disease. To the list of affections indicating a pathogenic heredity migraine should by all means be added.

The list of stigmata of degeneration (degeneracy?) and the directions as to examination of the gait, cranial nerves, reflexes, electric reactions, etc., should be of great service to those not accustomed to make a complete investigation of nervous cases.

A rather brief consideration of injuries to the brain, including a sketch of the more important localizations, opens Part I. Injuries to the spinal cord are treated somewhat more at length and with a clear-

ness that is admirable. Especially commendable appears to be the reasonable way in which the author handles the vexed question of spinal concussion.

All the necessary data involved in a diagnosis of lesions of peripheral nerves are clearly stated, including the somewhat confusing symptomatology of the various plexus paralyses.

In Chapter IV. the author has an exceedingly difficult task in estimating the ultimate organic effects of injury. He discusses the causative relation of injury to the psychoses, syringomyelia, epilepsy, tabes, general paresis, progressive muscular atrophy and paralysis agitans, and he acquits himself well. There is no doubt that most neurologists will approve of his conservative position on this question. The general tendency in the profession is to attribute too much importance to a precedent injury.

With Part II. begins the consideration of the traumatic neuroses proper, and the logical introduction (Chapter I.) is a concise and critical statement of the steps by which our knowledge of functional nervous disease following injury has developed. A comprehensive understanding of these diseases is scarcely possible without a knowledge of their historical evolution.

Passing next to the present, the author makes an admirably judicial statement of the present status of the traumatic neuroses, followed by a consideration of their causes and symptoms. In this whole chapter is clearly evidenced a strong grasp of the subject and a well-balanced judgment. To read it is a pleasure for the "Fachmann" and a profit for all.

Traumatic neurasthenia (Chapter II., 44 pages) and traumatic hysteria (Chapter III., 54 pages) receive separate and adequate attention. Although in individual cases a combination of the two diseases is exceedingly common, constituting what is quite properly called hysteroneurasthenia, yet the two affections are really separate entities, and a just appreciation of the combination must rest upon a clear conception of the integers. We quite agree with the author when he says that "as a distinct disease, hysteria is unfamiliar to most American physicians," and we are certain that the same may be affirmed of neurasthenia when caused by trauma—the largely surgical character of the cause seeming to cast a veil over the ordinary medical perspicacity. These two chapters, as well as the following one on unclassified forms, are heartily commended to him who wishes an understanding of the various forms of functional nervous trouble which often follow accidents, and it is devoutly hoped that their contents will ultimately constitute part of the thoroughly digested and well assimilated stock in trade of many a medical mind. The need of such healthful pabulum has already been emphasized.

The chapter on malingering is a thoughtful and pleasing essay, with the addition of a categorical enumeration of the different symptoms most frequently simulated, and the means of discriminating between fraudulent and bona-fide manifestations. In it the experienced and competent expert will find little that is new to him, but the physician who is unexpectedly called into a litigation case will find it invaluable. Even the neurologist whose time is devoted to private practice may be greatly puzzled by a case in which malingering is to be seriously considered, and may find many useful hints in the fifty-two pages of this chapter.

Closing the book are twenty-four pages covering the treatment of the traumatic neuroses, the greater part being, quite naturally,

devoted to the rest cure and hydropaths. They contain nothing new, but the means advocated are those approved by the best authorities and established by experience. The directions are sufficiently explicit.

As a whole, the work is decidedly good and should have a wide field of usefulness. The neurologist will probably not need to refer to it frequently, but the surgeon, the internist, the general practitioner and the physician whose work is in the more narrow fields of medicine, will here find a clear statement and well-balanced consideration of many things which they need to know. Although inevitably rather technical for a lay reader, the book should be of material aid to lawyers engaged in personal damage suits, and the writer ventures to believe that the ends of justice would be furthered were members of the judiciary not entirely ignorant of the facts and principles so well presented by Dr. Bailey. H. T. P.

THE PSYCHIATER. A Journal containing a Report of Scientific Work at the Illinois Eastern Hospital for the Insane. Published by the Medical Staff Quarterly. Hospital, Ill. 1898.

This, the first number of publication, would seem to be a modest attempt on the part of the authorities of the Illinois Eastern Hospital to give to the world some of the results of the work done along technical medical lines in that institution. The contents for the present number, *Professional Work in Hospitals for the Insane*, Report of Three Cases of Brain Tumor with Special Reference to the Pathology of Neuroglioma and the Psychical Changes Caused by Brain Tumors, The Early Diagnosis of Paretic Dementia and Laboratory Psychology as Applied to the Study of Insanity, are straightforward and simple contributions in their several fields. We wish a hearty success for the new journal.

THE MENTAL AFFECTIONS OF CHILDREN, IDIOCY, IMBECILITY AND INSANITY, by William W. Ireland, M.D. (J. and A. Churchill, London; J. Thin, Edinburgh, 1898.)

This book may be considered as an enlarged and revised work which has for its foundation the author's well known volume on Idiocy and Imbecility. As he states in his preface he here gives more matured views on the subject and has been enabled to bring together the widely scattered studies of able observers on the subject of idiocy and imbecility.

The work is largely devoted to a consideration of the idiot, for of the twenty-two chapters, at least eighteen of them are concerned with this class. In the opening four chapters, The Definition of Idiocy and Imbecility, Statistics of Idiocy, Causes of Idiocy and the Classification are treated. The author then describes in successive chapters the various types, genetous, microcephalic, hydrocephalic, eclampsic, epileptic, paralytic, traumatic, inflammatory, sclerotic and syphilitic idiocy, cretinism and the idiot by deprivation. The final chapters deal with the growth and mortality of idiots, insanity in children and insane idiots, sensory and mental deficiencies of idiots, the best methods of educating idiots and imbeciles, laws for idiots and imbeciles, while the last chapter touches upon the subject of "wolf boys."

The lucidity of the author's descriptions, the literary style and the mechanical excellence of the work will make it a standard for some years to come.

JELLIFFE.

BOOKS RECEIVED.

"Index Catalogue of the Library of the Surgeon-General's Office, U. S. Army." Second series, Vol. 3.

"Acromegaly," by Guy Hinsdale, M.D. Wm. Warren, Detroit, Mich.

"The Phonendoscope and its Practical Application," by Aurelio Bianchi, M.D. Geo. P. Pilling & Son, Phila.

"Transactions of the Iowa State Medical Society." Vol. XVI.

"A Text-Book of Obstetrics," by B. C. Hirst, M.D. W. B. Saunders, Phila.

"Thirty-first Annual Report of the Hudson River State Hospital," at Poughkeepsie, N. Y.

"Third Annual Report of the Rome State Custodial Asylum," at Rome, N. Y.

"Forty-eighth Annual Report of the Syracuse State Institution for Feeble-Minded Children," at Syracuse, N. Y.

Forty-third Annual Report of the Dayton State Hospital," at Dayton, Ohio.

"First Annual Report of the Boston Insane Hospital," at Boston, Mass.

"Thirty-eighth Annual Report of the Matteawan State Hospital," at Matteawan, N. Y.

"Annual Report of the Essex County Hospital for the Insane," at Newark, N. J.

"Annual Report of the Department for the Insane of the Pennsylvania Hospital," at Phila.

"Tenth Annual Session of the Association of American Anatomists," Washington, D. C.

"American Electro-Therapeutic Association, Eighth Annual Meeting," Buffalo, N. Y.

"Principal Poisonous Plants of the United States," by V. K. Chesnut. United States Department of Agriculture.

"The Second Hospital for the Insane of the State of Maryland," by Geo. H. Rohne.

"Primer of Psychology and Mental Disease," by C. B. Burr, M.D. F. A. Davis Co., Philadelphia, Pa.

"Wie ist die Fürsorge für Gemuthskranke von Aerzten und Laien zu fordern?" by Prof. C. Furstner. S. Karger, Berlin.

"Leitfaden der Electrodiagnostik und Electrotherapie für Praktiker und Studierende," by Dr. Toby Cohn. S. Karger, Berlin.

"Die Psychiatrische Klinik zu Giessen," by Dr. A. Dannemann. S. Karger, Berlin.

"The Principles and Practice of Medicine," by Wm. Osler, M.D. D. Appleton & Co., N. Y.

"The Sexual Instinct," by J. F. Scott, M.D. E. B. Treat & Co., N. Y.

"Jahresbericht über die Leistungen und Fortschritte auf dem Gebiete der Neurologie und Psychiatrie," von Drs. E. Flatau und L. Jacobsohn. S. Karger, Berlin.

"A Pocket Medical Dictionary," by Geo. M. Gould, M.D. P. Blakiston's Sons & Co., Phila.

"The Medical News Pocket Formulary for 1899," by E. Q. Thornton, M.D. Lea Bros. & Co., Phila.

"Saunders' Pocket Medical Formulary," by Wm. M. Powell, M.D. W. B. Saunders, Phila.

"Forty-third Annual Report of the Northampton Lunatic Hospital."

"Twenty-eighth Annual Report of the Central State Hospital of Virginia," at Petersburg, Va.

"Eleventh Annual Report of the St. Lawrence State Hospital."

"Biennial Report of the Alabama Bryce Insane Hospital," at Tuscaloosa.

"Chirurgie and Medicine," Ambroise Paré.

"Traite Pratique de Radiographie et de Radioscopie," par A. Londe, Paris.

"Sulle Alterazioni istologiche del Sistema nervosa centrale del Feto in Seguito and Avvelenamento acute e cronico della Madre per Alcool." Dr. M. Carrara, from "Rivista di Medicina Legale," 1898, No. 6.

"Lecons de Clinique therapeutique sur les Maladies du Systemé nerveux," par le Docteur Gilles de la Tourette. E. Plon, Nourrit et Cie., Paris, 1898.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

AN ACUTE MYXŒDEMATOUS CONDITION, WITH
TACHYCARDIA, GLYCOSURIA, MELÆNA, MANIA,
AND DEATH.*

BY WILLIAM OSLER, M.D.,

PROFESSOR OF MEDICINE, JOHNS HOPKINS UNIVERSITY.

Acute myxœdema may occur as a transient condition in goitre. In 1892, I reported the case of a young man, aged twenty-three, who had a goitre of moderate size, with which was associated for a period of five or six months a myxœdematous condition of the hands and face, which disappeared completely.

In 1893, I was consulted by Mrs. B., aged thirty-seven, who had exophthalmic goitre, and a swollen, myxœdematous state of the subcutaneous tissues of the legs below the knees. They did not pit; it was a brawny induration which had persisted for several months; there was no change in color. There are a good many observations in the literature of the co-existence of the two disorders, or of the development in myxœdema (sometimes following the use of thyroid gland extract) of the phenomena of Graves' disease, or vice versa. A brief summary of the recorded cases is found in Möbius' monograph in "Nothnagel's Specielle Pathologie und Therapie" (Vol. XXII, 1896).

The cases of Sollier¹ illustrate the usual sequence. A woman, aged thirty-one, seen first in March, 1891, had had

*Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

¹Sollier: *Revue de Méd.*, 1891.

exophthalmos in January, 1890, without enlargement of the thyroid, but with much nervousness, pallor, tachycardia, and well-marked tremor. There was in addition a very pronounced myxedematous swelling of the face, neck, and extremities, and supraclavicular fossæ. The thyroid gland was not enlarged, but seemed rather atrophied. In the second case, a woman, aged thirty-nine, who had had a good deal of mental worry and trouble, presented all the characteristic features of Graves' disease without goitre. The lobes of the gland could not be felt. The earliest symptoms were associated with rheumatic pains in the limbs and a transient œdema. There was much disability, and she was treated for chronic rheumatism. When admitted she presented the characteristic features of advanced myxœdema with enormous infiltration of the subcutaneous tissues. There was slight exophthalmos; pulse 110 to 120, and well-marked tremor.

I can find no description of a group of symptoms similar to that presented in the following record.

February 25, 1897. I saw to-day with Dr. Ellis, of Elkton, Mr. P., aged 31, an assistant freight manager on a western railroad.

Family history. There was no special tendency to nervous troubles. His father had been a dissipated man; his mother was living and well.

Personal history. He had enjoyed excellent health; had been very vigorous and strong. He was a man of exemplary habits; had not had syphilis, and had not been addicted to drink. He had been a very hard worker, and had been promoted rapidly to a very responsible position. He was married in 1892. He was a man of medium height, about five feet, eight, and his usual weight was 145 pounds. A photograph taken three or four years ago showed rather a thin-faced man.

Present illness. In October, 1896, his wife noticed that he was increasing rapidly in size, and before Christmas he had to get a completely new outfit of underclothes and outer garments. His weight, which as stated, was about 145 pounds, increased by January 1, 1897, to 182 pounds. He got very large in the abdomen, so much so that he suspected that he had dropsy, and in December remarkable scars appeared in the skin of the flanks. He felt pretty well and was able to attend to his work. His color was good, but every one remarked on the extraordinary increase in his size, and a personal friend asked him if he had been drinking, as he looked so bloated. He was at this time overworked, and his wife states that he became rather sleepless and irritable, and his usual disposition became changed. On and off, between

October and January, he had attacks of diarrhœa; the stools were sometimes dark colored, and he thinks there was blood in them. The movements were sometimes large and came on very abruptly, and once he had an almost involuntary evacuation. It is not altogether clear, however, that he actually at this time did pass blood. After the New Year he did not feel so well, complained of a good deal of prostration and weakness, and once he fell on the sidewalk from weakness. He kept at work, however, until February 5, when his friends insisted that he should go away for a change. He evidently could not at this time have felt very seriously ill, for he had a great deal of heavy business on hand, and worked up to three hours before leaving. He went to Florida, and while there became very much worse. He grew restless, wandered about a great deal, was sleepless, and got very "queer in his head." His wife said that he had certain delusions, said very funny things, and had an idea that people were troubling him. He said once that he would be all right "if he could get rid of these people." His skin had been very dry and harsh, and sometime in January, a red rash appeared on the upper part of the chest. In Florida he became so much worse, that he decided to return at once to Elkton, where his people lived. He arrived there on the 13th, and Dr. Ellis, who had known him from boyhood, states that he never was more shocked in his life than to see his condition. He was bloated; the face was almost purple, and he looked like a man who had been on a debauch for a month. He thought too that his eyes were a little prominent.

When I saw him on February 25, the patient was in bed, where he had remained since the 13th. He had improved in some ways, and Dr. Ellis thought his face had become very much less swollen. His mind had become perfectly clear, and he had no delusions. The features looked very heavy and bloated and congested; the lips were red, the cheeks flushed; the eyes looked a little prominent, the conjunctivæ were injected and watery. The eyelids covered the whites of the eyes; there was no Graefe's sign, no retraction of the lids, and the power of convergence was unimpaired. The tongue was slightly furred; the gums were natural looking. The neck looked thick and brawny; the supraclavicular pads were large and the lower part of the sternal notch was obliterated. Pulsation was noted in the carotids. The neck was flat in front, no prominence in the region of the thyroid, and the gland could not be felt.

On inspection of the thorax the skin looked congested and reddened in the upper part of the sternum, and there were the brownish scars of a rash in the upper part of the front of the chest.

The abdomen was full and large, and the skin presented in crescentic lines on either side in the flanks and in the iliac regions the most extraordinary atrophic lineæ, six on either side, the largest one extending in a curved line from near the tip of the tenth costal cartilage to within an inch of the spine of the pubes. It was fully three-fourths of an inch in breadth at its widest part. All were curved, and presented a purplish red color. The thighs and legs were large, but symmetrical. The skin looked everywhere dry, particularly on the backs of the hands and on the feet, and in the former situation looked infiltrated. While he was bloated and puffy, the general appearance was not at all that of a case of myxœdema.

There was not the slightest pallor or muddy hue of the skin. On palpation there was nowhere any tumor. The skin felt infiltrated and firm, and had to be picked up in large pieces, particularly over the backs of the hands and over the cheeks, everywhere a very solid infiltration. Over the manubrium and the lower cervical regions the infiltration was particularly marked.

The thyroid gland could not be well felt. If anything it was diminished in size. There was no enlargement in any of the groups of lymphatic glands.

When he arrived in Elkton, Dr. Ellis noticed the rapid action of the heart, and since then the pulse rate had not been under 120. The heart sounds were clear; there was no bruit at the base. The apex beat could be seen and felt a little outside the mammillary line. There seemed a little increase in the transverse area of dulness. Percussion over the manubrium was clear. The spleen was not enlarged. It was difficult to make a careful palpation of the liver. It was thought at first that perhaps the left lobe was enlarged, but on subsequent examination I think it was perhaps the serrations of the left rectus. Percussion gave no increase in the area of liver dulness.

The appetite had been good, and he had had no nausea, no vomiting.

After arriving in Elkton he had had on several occasions passages of blood, sometimes it was rather watery, no clots. He had sometimes as many as three and four stools in the day.

A special feature was the increasing weakness. Getting out of bed prostrated him very much, and he even had difficulty in sitting up, he felt so weak. There was a slight fine tremor of the fingers when the hands were held out, but I could not be certain that it was more than might be expected in a man who had become feeble and weak. There seemed no disturbance of sensation anywhere. The knee-jerks were present. From the time of his arrival in Elkton there had been

no sign of any mental disturbance. He seemed at times a little dull and apathetic, but the delusions had disappeared. He passed rather more urine than normal, but it contained neither albumin nor casts. His temperature was normal; during October and November his wife said that he constantly complained of feeling hot and flushed.

As I had seen this patient only at night, I visited him on March 1, in order to see the condition by daylight. The congested appearance of the face, the flushing of the skin of the chest on exposure, and the rapid pulse were very striking. On the other hand he had become apathetic and stolid. The eyes could scarcely be called prominent. The face looked very full and congested. The pulse had become more rapid, was 132 to 136, and occasionally dropped a beat. He had been sleeping very well at night, and he remained quite rational. Dr. Ellis thought that the weakness had increased considerably. He could no longer get up to use the commode. He had one involuntary passage. He had passed nearly eighty ounces of urine within fifteen or sixteen hours. The examination of it showed: deep yellow color, clear, no apparent precipitate; acid; 1.029; very large quantity of albumin; sugar present, reduces Fehling's and Mylander's solutions; polariscope, rays rotated to right indicating 2.5 per cent.; only a few finely granular casts, and a few squamous epithelial cells. There were still three or four stools in the day, usually thin and blood stained.

In a letter from Dr. Ellis, March 3, he states that the polyuria had persisted. The temperature had risen suddenly and had kept between 103° and 103.5° . He had become actively, even violently, delirious. The pulse continued with undiminished, indeed increasing, frequency. There was still blood in the stools. The most remarkable feature was the rapid diminution of the infiltration of the skin.

March 4. I saw the patient this morning. Dr. Ellis tells me that he began the thyroid extract on Monday, and continued it until Tuesday night, when the maniacal symptoms developed. He took in all twenty-five grains. Last night he had a combination of chloral and sulfonal and was much quieter, slept five or six hours. Throughout Tuesday night and the greater part of Wednesday he was in a very excited condition, using shocking language and making attempts to get out of bed, which he was really too weak to effect. The change in the patient since I saw him on Monday was very remarkable. He had become much thinner. The bloated infiltrated condition of the skin of the face and neck and upper part of the chest had lessened very greatly. There was not the same bloated aspect about the eyes, and the conjunctivæ

were not reddened. The abdomen, too, looked smaller, and there was evidently less infiltration about the legs and arms and hands. The skin was everywhere very dry and rough. He still looked flushed about the face and neck.

The pulse was between 140 and 145, regular, and of rather better volume than yesterday. The heart impulse was forcible, outside the nipple in fifth interspace. The pupils were of medium size, reacted to light.

The mental condition was peculiar. He seemed to recognize me. He was quiet most of the time; then would do odd things, as blowing three or four times forcibly, and frequently stretching out his hands to grasp imaginary objects, or he would ask some foolish irrelevant question. He was quite docile, and took food from Dr. Ellis; with the others he was a little obstinate. There was no jactitation and the tremor was very slightly perceptible. The diarrhœa had stopped for nearly thirty-six hours. The urine had been passed involuntarily. Examination of a sample by Dr. Fitcher showed the following : Specific gravity, 1.023, large amount of albumin, moderate number of fine and coarsely granular casts, and five per cent. of sugar.

On March 7, I received a note from Dr. Ellis, stating that the patient died of exhaustion that morning at nine o'clock. The active delirium never recurred. An autopsy could not be obtained.

Briefly summarized, a healthy man, weighing 145 pounds, rapidly increased in weight during three months to 182 pounds, the features became full and bloated, and the abdomen enlarged so rapidly that it split the corium in the inguinal regions in wide crescentic lines. Attacks of diarrhœa and marked irritability of temper were the only additional symptoms of moment. On February 5 he went South, and in Florida became extremely restless and had delusions. He returned to Elkton on February 13. From this date to March 7, the day of his death, his illness may be divided into two periods. To about March 1 the infiltrated, bloated condition persisted, his mind was clear, the pulse rate was not above 120, he had slight diarrhœa, sometimes with bloody stools. From March 1, coincident with the administration of the thyroid gland extract, he rapidly diminished in weight, and by the 4th he had lost in great part the bloated, infiltrated appearance. The tachycardia was more marked, he had become excited and delirious, and he had developed since March 1 an intense glycosuria.

The clinical picture presented by this case does not conform to any one disease, but presents certain combinations of myxedema with exophthalmic goitre. In the cases recorded

in the literature, so far as I can ascertain, the myxœdema has followed the symptoms of exophthalmic goitre at a variable period of months or years. This patient presented first the features of an acute, rapidly developing myxœdema. The increase in weight within three months was remarkable, but his appearance when I saw him first was not that of ordinary myxœdema. He had the bloated, swollen appearance of a stout man who had been drinking heavily. During the last part of his life the symptoms were those which we see in the toxæmia of acute exophthalmic goitre, viz, the tachycardia, the slight tremor, the delirium and the diarrhœa. When I saw him there was no evidence of exophthalmos, though Dr. Ellis thought that on his return to Elkton the eyes were a little prominent.

It seems most rational to suppose that in this case there was a perversion of the function of the thyroid gland, resulting in a toxæmia, which presented some of the features of myxœdema and some of Graves' disease.

DISCUSSION.

Dr. Booth said that in one of his cases—the one in which there was no improvement after thyroidectomy—the patient presented some of the symptoms which Dr. Osler had enumerated in the report of his case. There was a rapid increase in weight from one hundred and twenty-eight to one hundred and sixty pounds; the face was puffy, and the patient had frequent and severe attacks of diarrhœa, marked tremor, and a very rapid pulse, ranging from one hundred and forty to one hundred and sixty beats per minute.

Dr. Osler said that in the case he had reported there were some of the features of acute Graves' disease with mania; and that it was somewhat analogous to the case reported by Dr. Lloyd in which the disease lasted only a few days.

In those cases where sudden death follows the operation of thyroidectomy it would be interesting to learn whether there is any enlargement of the lymphatic glands and the thymus; it is possible that in such cases we may have a condition of so-called status lymphaticus, in which we know sudden death occurs.

NOTES ON THE TOXIC PROPERTIES OF THE BLOOD IN EPILEPSY.¹

By C. A. HERTER, M.D.,

VISITING PHYSICIAN TO THE CITY HOSPITAL, NEW YORK CITY, ETC.

Although the existence of a toxæmic element in the causation of epileptic seizures has repeatedly formed the leading topic of recent writers upon epilepsy there seem to have been no observations on the toxic properties of the blood of epileptic patients. With a view to obtaining some direct evidence as to the existence of such a toxæmic element I have made a number of experiments relative to the toxicity of the blood in epilepsy as measured by intravenous injections in rabbits. I shall refer briefly to the results of these observations in the hope that they may aid subsequent workers upon this difficult question.

The observations relate to the blood of fifteen epileptics at Craig Colony in the charge of Dr. Wm. P. Spratling and Dr. L. Pierce Clarke, to whose courtesy and cooperation I am indebted for the material and for the accompanying clinical histories. All of the fifteen patients have had grand mal seizures for several years. Owing to carelessness there is some uncertainty as to the identity of four of the patients whose blood was made the subject of study, and no attempt has been made to give histories of these cases. The method employed for determining the toxicity of the blood was that of intravenous infusion until the commencement of toxic symptoms. The rate of infusion was uniformly 5 cc. per minute. In four instances a second method was also employed, namely, that which consists in the intravenous injection of a much smaller amount of blood than is required to produce immediately toxic symptoms. Before speaking of the results of the infusions of the blood of our epileptic

¹Read before the American Neurological Association, May, 1898.

cases it is desirable to refer to the scope and limitations of the methods employed. Such reference is of the utmost importance, because there is frequently very great danger of misinterpreting the results of experimental injections of blood serum and of blood.

When normal human blood serum is continuously infused at the rate of 5cc. per minute into the femoral vein of a rabbit, fatal tonic clonic spasms begin when from 20-40 cc. per kilo have been injected. The same serum may be distinctly more toxic to one rabbit than to another of the same weight. There is reason to think that serum from the same normal individual may vary in its toxicity at different times. Moreover, it is not always possible to say whether a person whose serum has been taken as a normal serum is really quite a normal person at the time of venesection, for it is conceivable that the presence of temporary derangement of digestion may in some degree influence the toxicity of the blood.

There are reasons which seem conclusive for thinking that the toxicity of normal human serum for rabbits depends on a proteid substance in the blood. In many instances it is found that at the very beginning of the convulsive seizures there are coagula in the heart and pulmonary vessels. The toxicity of normal serum has been attributed by Haymen and others to this property of inducing intravenous coagulation, but in a small proportion of cases I have been unable to find any evidences whatever of coagulation immediately after spasm had begun. It is not yet clear whether a marked increase in the toxicity of the blood above normal is always due to an increase of the coagulation-inducing properties of the blood or whether the increase may depend on other toxic influences.² The rate

² It is now well known that the injection of an alien serum into the circulation of the rabbit may lead to the destruction of many red blood cells, with resulting hæmoglobinuria. Small quantities of serum do not cause sufficient destruction of red cells to cause hæmoglobinuria. In the observations referred to in the paper, hæmoglobinuria was not noted in any instance. In the case where rapid infusion was employed the animals lived much too short a time to develop hæmoglobinuria.

of infusion is a feature of considerable importance. In the observations recorded here the rate of 5 cc. per minute was employed. A slower rate, say of 2 cc. per minute, would have been preferable, as it would have required less blood to produce fatal symptoms. As already mentioned, the defibrinated blood was employed and not the serum of the blood. The difference in the toxicity of defibrinated blood and the corresponding serum probably depends entirely on the presence or absence of the red cells, the defibrinated blood being less toxic in proportion to the admixture with these cells. Thus the approximate serum toxicity of the blood may be estimated by regarding it as about 50 per cent. greater than that of defibrinated blood. Judged by this standard it is seen that of the fourteen cases in which the toxic values of the serum were determined by the method of rapid infusion these were normal in at least 12 instances. Of the two apparent exceptions one relates to a patient whose history was lost. The other exception relates to the case numbered 2, in which the seizures seem to be closely related to over-eating and are often associated with prolonged headaches, with congestion of the face and conjunctivæ. In this case the quantity of blood required to produce fatal symptoms was only 15 cc. to the kilo, which is equivalent to about 10 cc. of serum. This is a distinctly higher toxic value than that of normal serum. The observation just mentioned was made with blood drawn in the interval between two seizures. Another observation made about one month later, while the patient was suffering from a congestive headache following over-eating, gave no indication of increased toxicity of the blood, the values falling probably within normal limits.

In four instances an effort was made to determine the toxic values of the blood by means of a second method as well as by the one described. This method is also one of intravenous infusion, but instead of infusing until fatal

symptoms are initiated, an attempt is made to determine the minimum dose that will prove fatal in from 12 to 36 hours. This is the method generally employed in the study of the toxicity of blood serum. It is open to the objection that it may be necessary to make the infusion into several rabbits in order to make certain that the minimum quantity necessary to kill has been discovered, but is upon the whole a reliable method. The fatal dose of normal serum is usually regarded as varying from 8-12 cc. to the kilo of the animal's weight. Of the four trials referred to, the toxic values were apparently above the normal in two (Nos. 2 and 4). In one of these (No. 4) there is a want of correspondence between the result obtained by the first and that obtained by the second method, the value for the first method being well within normal limits. The other case is the one already referred to as characterized by congestive headaches. As with the first-named method, it is seen that there is an indication of exaggerated toxic properties of the blood during the interval between seizures. The quantity of the blood which killed a rabbit weighing 1800 gm., i. e., 10 cc., failed to kill a rabbit weighing 1750 gm. when taken from the patient during the congestive state. It is also to be noticed that on the occasion of the first trial with this patient's blood there was extensive clotting in the cavities of the heart and veins, whereas clotting was much less pronounced, or was entirely absent, on the occasion of the second trial.

In three instances the toxicity of the blood was tested immediately after a grand mal seizure, and then again after intervals of 15, 16½ and 26 hours respectively, in the three cases. In one case the blood appeared more toxic at the second bleeding than at the first, in another more toxic at the first bleeding than at the second, and in the third about equally toxic on the two occasions.

Two further observations deserve brief mention. In

one of these (No. 4) the blood taken three weeks after a seizure and two weeks before another was infused into a dog. The effects upon the animal were simply those which are observed from the infusion of normal blood in large volume.

The other observation was made with blood drawn 15 days after a seizure and several days before another. This blood was infused into the femoral vein of a Java monkey weighing 1200 gm. The quantity introduced was very large, 145 cc. The symptoms produced (mainly dyspnoea and cyanosis) were those which follow the infusion of a large volume of normal human blood into the circulation, and death was wholly referable to the mechanical action of the infused blood.

If we look over the data which form the basis of this report with a view to seeing what they teach us about the toxic properties of the blood in epilepsy, it is clear that no positive conclusions can be reached. In all of our cases, except one, the evidence indicates that the toxicity of the blood was not increased, or that the results of different infusions are contradictory. The criticism might be made that the blood of certain of our epileptic patients may have contained toxic substances whose presence would not be indicated by the use of so crude a method as that employed. I am disposed to think that such a criticism would be entirely just. Although the method suffices to indicate that in pneumonia and uræmia the toxicity of the blood may be markedly increased, it is probable that it would fail to indicate slight variations from the normal toxicity. Hence it can only be said that the evidence, such as it is, does not indicate any increase in the toxic properties of the blood of most cases of epilepsy, either immediately after the seizures or in the intervals.

As regards the one case in the series in which the blood appears to have been distinctly more toxic than normal in

an interval between the paroxysms, no positive conclusion can be reached. It is possible that this increase in the toxic action of the blood may be in some manner connected with the disorders of digestion, dependent on indiscretions in diet, which are characteristic of this patient. It is possible that the blood of normal persons may temporarily be increased in toxicity under similar conditions of disordered digestion.

In conclusion, it may be said that the chief purpose of this report is to call attention to the very great difficulties that attend the investigation of the toxic properties of the blood in epilepsy. These difficulties are largely the result of the crude nature of the available methods of study, which are poorly adapted for the detection of slight differences in the toxic values of blood, although they have given important results in the investigation of the well-defined toxæmias of pneumonia, uræmia and scarlet fever. Those shortcomings may, however, be in some degree lessened by the use of certain precautions in using known methods, and the following suggestions are made for the consideration of those who undertake further researches upon the toxicity of the blood of epileptics:

First: In testing the toxicity of the blood by rapid and continuous infusion into the circulation of rabbits the rate of infusion may advantageously be limited to 2 cc. per minute, and the infusion should be stopped when the first indications of involuntary spasm appear. It is desirable that the weight of the rabbits employed should not vary beyond certain fixed limits—say 1400-1600 gm., and that all the animals should be of the same breed.

Secondly: The method just mentioned should be controlled by means of the method which has for its object the determination of the minimum intravenous injection that causes death in from 12 to 36 hours. At least three observations should be made by this method for each

Name.	Rate. c.c.	Quantity. c.c.	No. of c.c. to Kilo.	Weight of Rabbit.	Symptoms.	Remarks.
No. 1—C. K. Bled 3.30 p.m. Dinner at noon.	5	48 8	32 5.25	1500 1520	Contracted pupils, tonic clonic spasms. Recovered.	
No. 2—C. W. Bled 3 p.m. Dinner at noon. Dec. 30, '97.	5	26 10	15 5.50	1740 1800	15 c.c., pupils contracted. 20 c.c., cyanosis and dyspnoea. 26 c.c., tonic clonic fibrillation. Drowsiness in a few minutes. Died in 45 minutes in spasms.	Extensive clotting in veins and heart.
Jan. 28, '98. In congestive state. Dinner at noon. 3 p.m.	5	95	55	1710	5 c.c., pupils slightly contracted. 25 c.c., twitching face and nose. 80 c.c., eyeballs prominent. 95 c.c., severe tonic clonic spasm.	Moderate clotting.
	5	52 10	32 6	1650 1750	20 c.c., pupils contracted. 52 c.c., spasm and death. Recovered.	No clotting in heart.
No. 3—W. D. M. 3 p.m. Dinner at noon.	5	35 15	30 11	1180 1335	5 c.c., contracted pupils. 35 c.c., tonic clonic spasms. Died in 40 minutes. Slight spasm in legs.	Extensive clotting in four chambers of heart and pulmonary veins.
No. 4—W. H. J.	5	70 20	37.68 9.32	1845 2145	Pupils slightly contracted. No spastic symptoms. Died in 15 or 20 minutes. Very drowsy. No spasms.	No thrombi of heart or vessels. No thrombi in heart or vessels.
8 days after seizure.		50	24.75	2020	Dyspnoea and cyanosis.	General clotting.
10 days after seizure.		45	40.72	1105	Dyspnoea and cyanosis.	General clotting.

3 weeks after seizure. "	5	40 110	31.19	1205 Dog 10 k.	Severe spasms and death. Breathing slow and deep. Lived 48 hours.	
4 weeks after seizure.		20	14.5	1380	Small pupils. Rapid breathing. Lived 24 hours.	
No. 5—A. H. No seizure for 6 days.	5	68	43.09	1578	Contracted pupils at 50.	Extensive clotting.
No. 6—A. H. No seizure in 4 days.	5	45	25.14	1790	25 c.c., contracted pupils.	Clotting.
No. 7—N. J. No seizure for 7 days.			22.77	1537	Died with usual symptoms, 35 c.c.	
No. 8—B. C. No seizure for 15 days.	5	45 145	51.42	875 Java monkey 1200	Contracted pupils. Drooping lids—after 100, cyano- sis. No spasm at any time. No clotting. Patches of œde- ma and congestion in lungs. No congestion of intestine.	
No. 9—A. W. Grand mal seizure 2.45 p.m. Bled 3.30 p.m. 15 hours after seizure.	5	105	36.2	2900	Usual symptoms. Symptoms begin at 50.	
No. 10—C. S. Seizure 6.55 p.m. Bled 7.35 p.m. 26 hours later.	5 5 5	100 90 50 38	52.63 50 27.77 20.76	1900 1800 1800 1830	70 c.c., pupils slightly contracted. Fibrillation marked.	
No. 11—K. A. Seizure 4.30 p.m. Bled 5.15 p.m. " 16½ hours after seizure.	5 5	90 65	43.9 46.42	2050 1400	Symptoms at 60. Symptoms as above.	

sample of blood, and the average of the toxic values should be taken. The results may be depended on if there is an agreement between the indications of the methods.

Thirdly: When practicable the toxicity of the serum for guinea pigs should be determined by subcutaneous injection, as recently suggested by Uhlenhuth, working with Brieger. This method has the advantage of obviating the effects of intravenous coagulation.

Fourthly: Should there be cases where the toxicity of the blood appears regularly to be increased near the time of seizures it would be well to make a further test by intravenous infusion into monkeys. In these animals intravenous coagulation seems not to be induced by human blood. Moreover, this animal's means of expression are so perfected that slight toxic effects appear to be more readily discernible in him than in other animal types.

To carry out studies as thus suggested is laborious, and requires the use of large quantities of blood, but I am convinced that reliable physiological evidence as to the toxic properties of the blood in epilepsy cannot be obtained except through laborious and numerous observations. It is likely that some chemical alterations in the blood, not without pathological significance to the organism, could be entirely overlooked even with the use of the most painstaking methods of physiological inquiry.

No. I.—C. K., 32. Occupation, farmer. Paternal grandmother died of phthisis. Two sisters died of kidney disease (?). His epilepsy began at the age of 29; supposed to have been due to rheumatism, patient having suffered from an attack of acute articular rheumatism and pericarditis some two years prior to his first epileptic fit. About one half of his attacks are grand mal in character; the others are of psychic type. At the time of admission to the Colony, patient suffered from temporary glycosuria, which lasted for several weeks. Albumin in small quantities was also present in urine. This condition rapidly disappeared after having been placed upon anti-diabetic diet. Although patient has had frequent severe attacks since absence of sugar in the urine was noted, yet he has never had a return of this functional phenomenon.

No. 2.—C. W., 18. Occupation, housework. Father has at times been intemperate. Mother died of phthisis. Epilepsy began in infancy and was supposed to have been caused by "heart disease." Attacks are almost all grand mal in character, occurring by day, and are preceded by a feeling of dizziness and pain in the epigastrium. Heart shows mitral regurgitation. Patient has an abnormally large appetite and has frequently eaten inordinate quantities of food and vomited on several occasions soon after. At times preceding her attacks, and occasionally at other times when no attacks follow, she has suffered from prolonged congestive headaches. The face is red and the conjunctivæ are congested; the face bears the appearance of one coming from the hot room of a Turkish bath. At times patient has been placed upon restricted diet and reduced some twenty or thirty pounds in weight, with marked improvement in her seizures and entire abolition of her congestive headaches. Whenever she is allowed to return to the regular dietary she immediately regains her former stout physical condition and her seizures return to their former frequency and severity.

No. 3.—W. M., 40. Occupation, merchant. Patient's relatives for several generations have at times been somnambolic. Maternal grandmother died of epilepsy. Patient's father died of brain fever. Epilepsy began at the age of 34. It is supposed to have been caused by prolonged dissipation and generally dissolute life. Attacks are both grand mal and petit mal. Patient is frequently automatic after both kinds of attacks and is occasionally violent for several days. Attacks are all preceded by epigastric aura. He has suffered from considerable mental deterioration since his epilepsy began. Frequently, prior to his attacks for two or three days, his face is markedly congested, conjunctiva injected, and pulse is hard, wiry and increased in frequency.

No. 4.—W. H. J., 25. Occupation, farmer. Mother died of cancer of the breast. Patient's epilepsy began at the age of 18 and is supposed to have been caused by an injury to the head by a tree falling upon him about six months prior to first fit. Attacks are almost all grand mal in character. For the past two years, since patient's admission to the Colony, he has suffered from periodic attacks of mental stupor, in which his general appearance is that of melancholia cum stupore or acute primary dementia. In these states, which are not at all regular, patient loses greatly in bodily weight and bed sores frequently form in various parts of the body, apparently as a result of pressure and mal-nutrition. One examination of the blood conducted in this case was made between his periods of stupor when he was about the Colony daily at work.

No. 5.—A. H., 27. Occupation, seamstress. General neurotic history among paternal relatives. Maternal grandfather markedly intemperate. A younger brother is feeble-minded. Epilepsy began at the age of 14. Her mother suffered from considerable mental stress just prior to the patient's birth, and the labor was very difficult and performed by the aid of instruments. No cause was assigned for her epilepsy. Attacks at first were petit mal, but are now grand mal in character; they are frequently followed by automatism and occasionally considerable mental disturbance precedes and accompanies a series of attacks. No aura. Patient is quite feeble-minded.

No. 6.—A. H., 24. Occupation, housework. Mother has had severe headaches for years, and father has had rheumatism the greater part of his life. Patient's epilepsy began at the age of 14. No cause was assigned. Attacks are almost all grand mal in character and usually occur at night, preceded by no aura and followed by marked mental depression. Considerable mental deterioration has taken place. Heart, mitral regurgitant murmur. Knee-jerks atypically active. Several times since patient's admission to the Colony she has suffered from temporary eczema of either the right or the left hand, which condition immediately improved under topical treatment and proper management of the dietary, especially when nitrogenous food was withdrawn.

No. 7.—N. J., 24. Occupation, housework. No neurotic history obtainable. Epilepsy began at the age of 12. Assigned cause of epilepsy was overstudy at school. First attack occurred at the first menstruation, which appeared to be normal. Her attacks are almost all grand mal in character and are preceded by no aura. Patient is in very good mental health. She very frequently suffers from acute exacerbation of chronic eczema, which is quickly controlled and removed by proper attention to the dietary, and especially when placed upon milk diet exclusively.

No. 8.—B. C., 28. Occupation, housework. Patient's father died of paralysis (?). Mother died of cancer of the uterus. Patient's epilepsy began at the age of 17; cause ascribed to mental worry and an overdose of toothache medicine. Attacks are almost all grand mal in character and generally occur in series, preceded by an aura consisting of a sensation of numbness in the fingers of the left hand. At times patient has led a variable life and has been subject to sexual excesses since epilepsy began. Reflexes uniformly atypically active. Patient almost always suffers from marked mental depression following her seizures and considerable disorder in alimentation.

No. 9.—A. W., 24. Occupation, housework; epilepsy existent since 12 years of age, although patient had quite a number of eclamptic convulsions at dentition. No neurotic family history was obtainable, except that the father was very intemperate and died from alcoholic excesses. At the age of 10, patient had cerebral palsy, leaving left side hemiplegic. The supposed cause of epilepsy was this hemiplegia. Aura, mental confusion. Mind somewhat enfeebled. All reflexes of left side are markedly exaggerated. Almost all attacks are grand mal.

No. 10.—C. S., 27. Occupation, farmer. No neurotic history obtainable. Epilepsy supposed to have begun at age of 17, although patient had many convulsions at dentition. Supposed cause, abnormal dentition. Almost all attacks are grand mal. Aura, muscular trepidation in the calves of both legs for two or three minutes prior to convulsion. Patient is in very fair mental condition. He has frequently complained of severe and prolonged headaches and general mental depression some days prior to his convulsions.

No. 11.—K. A., 32. Occupation, housework. No neurotic history obtainable. Epilepsy began at the age of 13. Supposed cause, severe fright. Convulsions are almost all of grand mal type and of the most violent kind, the tonic and clonic stage often lasting for five or ten minutes. Mind somewhat weak. Patient is often very irritable just before and after seizures. Knee-jerks uniformly atypically active. Pupils dilated.

20. EIN FALL VON PROGRESSIVER PARALYSE BEI MUTTER UND KIND (A Case of General Paresis in Mother and Child). O. Müller (*Allgemeine Zeitschrift f. Psychiatrie*, 55, 1898, p. 151).

The author here presents the clinical and pathological histories of two cases of general paresis occurring in a child of eleven, and in the mother at the age for forty-five.

There was a neurotic family history, and the mother had syphilis. One younger child died at an early age, of convulsions, said to have been caused by congenital syphilis. The mental affection first was present in the daughter, who up to the age of ten or twelve was a promising young girl. The first symptoms noted were those of inability to skate well, the girl having been quite an adept in this pastime. The writing soon became affected, and the girl commenced to be left behind in her classes as a result of her gradually increasing dulness which advanced to a complete and helpless dementia. The mother began to be affected a few years later than the daughter. Her first symptoms were of the nature of extreme jealousy. Later she became very indolent and careless of her person, began to drink freely, and unmindful of her household duties. Paresis of the muscles of articulation was an early symptom, and the disease progressed in typical fashion.

The post mortem examinations showed the generally received lesions of general paresis in both cases.

A CASE OF AMYOTROPHIC LATERAL SCLEROSIS PRESENTING BULBAR SYMPTOMS, WITH NECROPSY AND MICROSCOPICAL EXAMINATION.*

By F. X. DERCUM, M.D.,

CLINICAL PROFESSOR OF NERVOUS DISEASES, JEFFERSON MEDICAL COLLEGE;
NEUROLOGIST TO THE PHILADELPHIA HOSPITAL, AND

WILLIAM G. SPILLER, M.D.,

PROFESSOR OF DISEASES OF THE NERVOUS SYSTEM IN THE PHILADELPHIA
POLYCLINIC; ASSOCIATE IN THE WILLIAM PEPPER CLINICAL
LABORATORY, UNIVERSITY OF PENNSYLVANIA.

The cases of amyotrophic lateral sclerosis studied clinically and pathologically have not been very numerous, and the report of every case increases our knowledge of the disease. Wolff¹, writing in 1894, stated that though amyotrophic lateral sclerosis had been known for about thirty-three years, he had been able to collect only ninety cases from the German, French, English and Russian literature, and only fifty-six with necropsy. Collins² succeeded in finding seventy-two cases with necropsy in 1896. We hope, therefore, that the following case may be of interest.

C. K.; a male; a native of Ireland; a laborer; married, aged fifty-three years, was admitted to the Nervous Wards of the Philadelphia Hospital, June 14, 1892. His family history was negative as regards nervous diseases. His personal history was unimportant up to the beginning of the present affection. He denied venereal disease and alcoholic excesses.

For five or six months before admission he had complained of pains in both legs from the knee down. He would grow very tired by night, and was soon obliged to use a cane in walking. Rest in bed would generally relieve him so that in the morning his legs would feel all right. On admission to the hospital the legs were somewhat spastic with exaggerated reflexes. Cutaneous sensibility was not changed. He walked with crutches and regularly left the hospital in the summer season for a period of two months. One year after admission his feet began to swell, and this œdema recurred from time to

* Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

¹ Wolff: *Zeitschrift für klin. Med.*, xxv., p. 326.

² Collins: *Amer. Jour. of the Med. Sciences*, cxi., 1896, p. 690.

time. Later, *eczema rubrum* of both legs developed. In the summer of 1895 he was unable to leave the hospital as before, the weakness and rigidity of the legs now necessitating a wheel chair. About this time also it was noted that his speech was becoming indistinct, and it gradually assumed a bulbar character; but long before this he stated he had lost the power of whistling.

The arms also gradually became spastic; the exact time at which these symptoms made their appearance is not known. It appears; however, to be beyond question that the involvement of the arms was not only subsequent to that of the legs, but also subsequent by several years, as spasticity of the arms is not mentioned in the earlier notes.

All of his symptoms gradually became more pronounced until April, 1897, when he became bed-ridden, had occasional retention of urine and loss of control over the bowel, and developed bed-sores over the sacrum, heels and elbows. Subsequently his general condition again somewhat improved.

At the examination on November 11, 1897, the following notes were made: He is found seated in a wheel chair. His arms are semiflexed, the position of the limbs suggesting lateral sclerosis. Voluntary movements are much restricted and exceedingly slow, and only very slight flexion of the thighs or legs is possible, while the feet are in extreme extension. Marked resistance is also offered to passive movements, especially at the knees and ankles. The muscles feel firm, but some general wasting of the legs appears to have taken place. The knee-jerks are much increased.

The voluntary movements of the arms are also much restricted, but less so than those of the legs. The arms cannot be extended at the shoulders to the normal degree, but the amount of movement is considerable. Much less movement is possible at the elbow, and still less at the wrist and fingers, contracture being most marked in these situations. The fingers can be extended slowly and with difficulty. Considerable resistance is everywhere offered to passive movements. General wasting is noted in both arms, more marked in the forearms. Wasting is also noted in both hands; the thenar and hypothenar eminences are somewhat flattened. All of the symptoms are slightly more pronounced in the right arm than in the left. The tendon reactions are all increased.

The head can be moved voluntarily in all directions, but the movements are somewhat slower than normal and slightly irregular and jerky. The patient sits with the head slightly flexed, and does not move it unless requested to do so. However, the movements of the head are much freer on the whole

than the movements of the arms and legs. No resistance is offered by the neck muscles to passive movements.

The face presents no asymmetry. The mouth is held slightly open. When he is asked to show the teeth, the mouth is drawn very slightly to the right. He is able to compress the lips fairly well. He cannot whistle; on making the attempt the lips deviate slightly to the right, while the opening formed between them is large and somewhat irregular. No tremor of the lips is observed, but there is unmistakable irregularity or ataxia of movement while making the effort. There is no dribbling of saliva. The muscles of expression appear to be normal. When he is asked to protrude the tongue, the whole mass is protruded sluggishly and it is launched forward with evident effort and toward the right side. Fibrillary twitchings are evident on both sides of the tongue. The tongue as a whole seems slightly atrophied; the right half being slightly smaller than the left. Swallowing is performed with difficulty, though there is no regurgitation through the nose. In eating solid food the patient assists the tongue by pushing the food between his teeth with his fingers.

The patient talks with a nasal intonation as though the soft palate were paretic. Only faint movements of the soft palate are seen during phonation. The speech is drawling and indistinct, and the consonants are imperfectly formed. The voice appears to be slightly hoarse and its pitch lower than normal. (The larynx, unfortunately, was not examined.) The pupils react normally to light and in accommodation. The movements of the globes are normal in all directions.

There is some atrophy of the muscles of the shoulder girdle, but it is not marked. No noticeable changes are observed in the trunk, save that the chest is somewhat barreled. There is retention of urine and incontinence of feces.

No sensory losses can be detected. Touch, temperature and pain sense appear to be normal. The only subjective symptom is occasional pain in the knees.

The patient's mental condition appears to be fairly normal, save that he manifests a marked tendency to causeless laughter and that his memory is not as good as formerly. The general visceral examination is negative. A small bed-sore is found over the sacrum.

Subsequently the patient's general condition again became worse. Complete paralysis of the sphincters and great general prostration gradually made their appearance. December 30th he suffered greatly from abdominal pain and passed large quantities of bloody urine. His condition became gradually worse day by day until finally respiration became labored, circulation failed. He died January 7th, 1898.

Necropsy.—The body is that of a well nourished male. Dark coppery discoloration of both feet is noticed, extending midway up the legs. The toes are very strongly flexed and the sole of the foot is markedly arched. A condition of equinovarus exists. Lividity of surface is noted posteriorly. The hands and arms are extended.

Abdominal Cavity.—On opening the abdomen the general appearance is normal, but on close examination a fold of intestine is found bound to the top of the bladder by recent adhesions. On tearing these free a gangrenous perforation of the top of the bladder is noted. This opening was entirely walled off by two loops of the sigmoid flexure of the colon. A thick layer of lymph is upon the bowel where it was adherent.

Thoracic Cavity.—No plural adhesions are found and no effusion in either sac.

Heart.—Pericardium is normal, but the heart muscle presents evidences of slight myocarditis. A few patches of beginning atheroma are found in the aorta.

Lungs.—The lungs are crepitant throughout and slightly emphysematous. The arteries are sclerotic.

Spleen.—Spleen is normal.

Left Kidney.—The suprarenal body is normal. The capsule of the kidney strips easily. The perirenal fat is much injected. The surface of the kidney is stippled with small white spots, the size of a pin's head. These are barely perceptible to touch. They seem to be small miliary abscesses. On section, a quantity of foul-smelling fluid ran out. Two or three stones are found in the substance of the kidney. The largest is about the size and shape of a jackstone. The cortex is of good size and injected. The pelvis is distended and much inflamed.

Right Kidney.—The suprarenal body is normal. The kidney has a cyst the size of a walnut, and several smaller ones are seen on section. It is much like its fellow except that it does not seem to be so much diseased. The pelvis is not dilated and contains no stones. The capsule strips easily.

Ureters and Bladder.—The bladder is small, ribbed, thickened, and has a diphtheritic-looking membrane covering its inner surface. The edge of the rupture is ragged and easily torn.

Liver.—The liver is normal. The gall-bladder contains a small quantity of thin yellowish bile, and one small mulberry calculus. The duct does not seem to be patulous, or at least no bile can be forced through.

The brain and spinal cord presented no gross abnormalities and were reserved for microscopical examination.

Microscopical Examination.—In the lumbar and cervical regions the anterior roots are normal, or very slightly altered

when stained by ammonium carmine or Weigert's hæmatoxylin method, although the cells of the anterior horns are few in number, especially in the inner and posterior groups. Most of these cells when stained by thionin according to v.

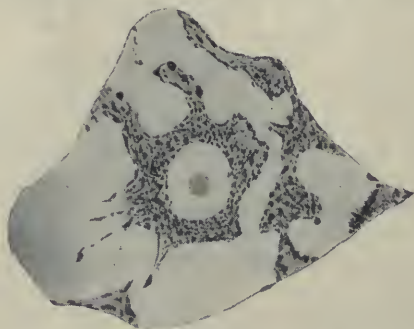


FIG. I. An anterior horn cell in which the chromophilic elements have undergone a peculiar form of degeneration.

Lenhossék's method have processes and contain chromophilic elements and centrally situated nuclei. Many of the cells are greatly atrophied. The cells contain also a large amount of

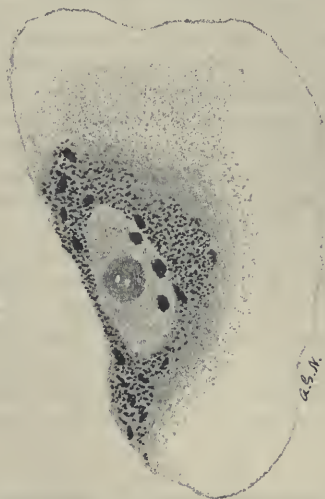


FIG. II. An anterior horn cell presenting a high degree of peripheral chromatolysis.

pigmentary substance, and occasionally a mass of so-called pigment is all that remains of a former cell. Complete chromatolysis, either central or peripheral, is not found in most of the cells, though in some of the smallest cells the chromophilic

elements are broken into granules and no longer appear as elongated bodies. Occasionally transverse cleavage of the cell body may be seen. We have only been able to find one or two cells in which peripheral chromatolysis is very complete. In one cell we have noted the existence of what appears to be two nuclei. The atrophy of the cells of the anterior horns is much greater than that of the anterior spinal roots. The anterior horns throughout the cord, both by the carmine and Weigert's hæmatoxylin stain, are much less intensely colored than the posterior, and have a rarefied appearance. They contain comparatively few fibres. The posterior roots are normal.

The crossed and direct pyramidal tracts are much degen-

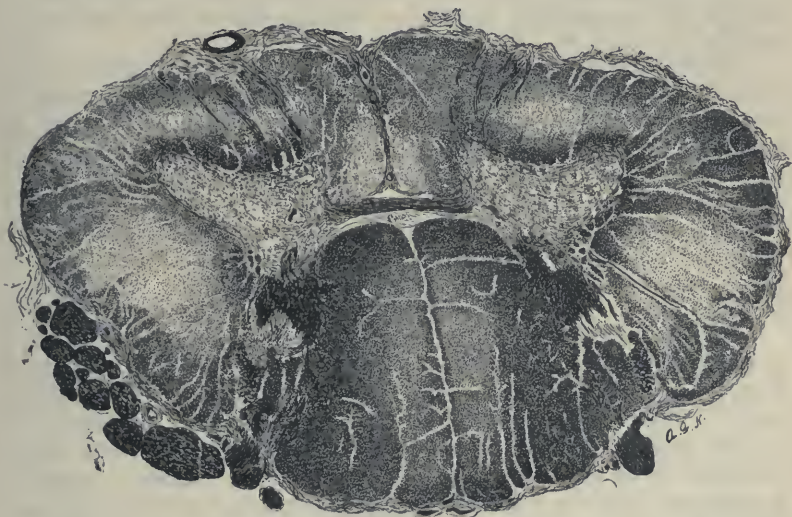


FIG. III. A section from the lower cervical region. The antero-lateral columns, especially the pyramidal tracts, are much degenerated. The posterior columns are slightly degenerated. The anterior horns are less intensely stained than the posterior.

erated in the spinal cord, and yet they contain many normal fibres. The sclerosis involves a much larger portion of the anterolateral columns than the area occupied by the pyramidal fibres. The posterior columns in the lower cervical and upper thoracic region present a sclerosis which does not extend to the posterior commissure, posterior horns, or periphery of these columns. This sclerosis is not confined to any system of fibres and is only of moderate degree. The Marchi method reveals no recent degeneration. The degeneration of the pyramidal fibres is very distinct at the motor decussation, but above this becomes gradually indistinct, though the anterior pyra-

mids are very evidently diseased. The cells of Clarke's columns are apparently normal.

A portion of the external popliteal nerve was "teased" and placed in the fresh state in osmic acid. The fibres present some swelling of the myelin sheaths, but otherwise they are normal.

The facial and hypoglossal centres and the nucleus ambiguus are normal, though some of the cells are highly pigmented. The posterior nucleus of the vagus contains many cells showing great pigmentary degeneration, and masses of so-called pigment in some places are the only remains of nerve cells. Intramedullary portions of the vagus and hypoglossus nerves are apparently normal. The posterior longitudinal bundles are normal.

Round bodies are found immediately under the fourth ventricle at the level of the vagus and hypoglossus centres, and they are also found within the spinal cord. They are round, of various sizes, and when freshly stained seem to be surrounded by a clear space, but after the section has been kept for a time these bodies present a pale blue peripheral circle. These formations are the same whether the tissue is cut transversely or longitudinally. They stain a deep blue with thionin and retain the color very well, and are also colored by Delafield's hæmatoxylin. They are doubtless amyloid bodies.

Groups of round bodies are found in the medulla oblongata, especially about the vessels. They stain deep purple with thionin, but fade very soon to pink, and gradually lose all color. They may possibly be the colloid bodies of Bevan Lewis, and have been described by us³ elsewhere. They do not seem to be the same as the amyloid bodies. When thionin is used and Lugol's solution applied afterward, the nerve cells, neuroglia cells and so-called colloid bodies stain a light mahogany color. The amyloid bodies have the same deep mahogany color as when the Lugol's solution is used alone.

Portions of one biceps muscle of the thigh appear to be normal. We were not able to remove any tissue from distal portions of the extremities. The tongue studied by the Marchi stain and by carmine is normal, and even its intramuscular nerve fibres are not diseased. Interstitial myositis is found in sections taken from one crico-arytænoides posticus muscle of the larynx. The connective tissue here is hyperplastic and the nuclei are excessive. In some places areas of degenerated muscular fibres are found.

The giant cells of the paracentral lobule are not destroyed, but are not abundant. Numerous pieces of tissue were taken

³ Spiller: New York Medical Journal, Aug. 13th, 1898.

from different parts of each lobule, but only a few giant cells were observed. With this exception, the cortex seems to be normal.

Portions of one paracentral lobule, hardened in formalin and stained by Marchi's method, reveal no traces of degeneration. Sections from the inner capsule, hardened in Müller's fluid and stained by the same method, give negative findings. Sections from the pons, cerebral peduncles and inner capsule reveal no degeneration by Weigert's method.

The columns of Goll are not infrequently affected when the pyramidal tracts are diseased, especially if the latter are primarily affected, and the columns of Goll in such cases stain more deeply with the carmine. This was the condition in a case of arrested development reported by one of us (Spiller⁴). According to Brissaud the columns of Goll are the most liable of all the spinal columns to degenerate.

Dejerine and Sottas⁵ describe a case in which they found sclerosis of the crossed pyramidal tracts, not extending upward above the superior part of the cord. No appreciable change could be detected in the cells of the anterior horns, or in the anterior roots, and no muscular atrophy had been seen. In this case the columns of Goll were slightly sclerosed in the cervical and upper thoracic regions. Sensation had been normal during life. This case might possibly be regarded as one of amyotrophic lateral sclerosis in its early stages, beginning in the pyramidal tracts, and in this sense it would support the view of Charcot regarding the portion of the nervous system primarily affected in this disease. We are not warranted, however, in assuming that the peripheral neurons would necessarily have become involved.

Moeli⁶ observed degeneration of the posterior columns in the lower cervical and upper thoracic regions in a case of amyotrophic lateral sclerosis.

⁴ Spiller: *Journal of Nervous and Mental Disease*, Feb., 1898, p. 87.

⁵ Dejerine and Sottas: *Arch. de Physiologie*, July, 1896.

⁶ Moeli: *Archiv. für Psychiatrie*, x., p. 718.

Marie⁷ says that the columns of Goll in amyotrophic lateral sclerosis stain more deeply with the carmine than they do in normal spinal cords. He attributes this to alteration of the myelin sheaths, and probably to overgrowth of the neuroglia, which may be the result of lesions of the cells of the posterior columns situated in the spinal cinerea. Inasmuch as Lenhossék⁸ finds that few cells of the cord send their axis cylinders into the posterior columns, we must be in doubt as to the correctness of this explanation.

Not infrequently the columns of Goll present a slight sclerosis in cases in which no sensory symptoms have been present, and there seems to be a relation in some way between this sclerosis of the columns of Goll and that of the motor system. Oppenheim⁹, and Charcot and Marie¹⁰ noticed sclerosis of the posterior columns of the cord in amyotrophic lateral sclerosis. Other cases are reported in which symptoms indicating involvement of the posterior columns were added to those indicating disease of long duration of the lateral columns. Brissaud¹¹ reports such a case. At the necropsy the lesions of amyotrophic lateral sclerosis and of incipient tabes were observed.

Degeneration of the posterior columns has been found in chronic anterior poliomyelitis. Nonne¹², in reporting a case of this disease, says he noticed a distinct, though not excessive, rarefaction of fibres in the median portion of the posterior columns. This was most evident in the cervical region, and was not limited to any system of fibres. The cells of the anterior horns were intensely degenerated. Inasmuch as primary degeneration of the pyramidal tracts was not observed, it seems reasonable to

⁷ Marie: *Traité de Médecine*, vi., p. 347.

⁸ Lenhossék: *Der feinere Bau des Nervensystems*, etc.

⁹ Oppenheim: *Archiv. für Psychiatrie*, xxiv., p. 758.

¹⁰ Charcot and Marie: *Archives de Neurologie*, x., 1885, pp. 1 and 168.

¹¹ Brissaud: *Leçons sur les Maladies Nerveuses*, p. 34.

¹² Nonne: *Berliner klin., Wochenschrift*, 1896, p. 207.

ascribe this degeneration of the posterior columns to the destruction of the cells in the spinal cinerea, and not to a primary degeneration of the nerve fibres. Experiments performed on animals in temporary closure of the abdominal aorta have given support to Marie's view and have shown that many cells within the spinal cinerea send their axis cylinders into the posterior columns.

Hektoen¹³ described a case of amyotrophic lateral sclerosis in which sensory symptoms were noted later in the disease. He ascribed these symptoms to the lesions of the spinal ganglia, posterior nerve roots, columns of Goll, and the sensory portion of the fifth nerve. He regarded his case as a sort of connecting link between progressive spinal muscular atrophy and amyotrophic lateral sclerosis on the one hand, and *tabes dorsalis* on the other.

A sufficient number of cases of combined systemic disease have been reported to show us that tabetic symptoms may appear at the same time as, or later than, those indicating disease of the lateral columns, but in cases of amyotrophic lateral sclerosis in which symptoms pointing to an affection of the posterior columns have not existed, the sclerosis of the columns of Goll is not always tabetic in its location within these columns, i. e., it is not systemic in character and is unlike that which we find with disease of the posterior roots. In our case the posterior roots are not degenerated, and the more deeply stained portion of the columns of Goll, by the carmine stain, is found in the lower cervical and upper thoracic regions, and does not extend to the posterior commissure or to the periphery of the cord. It is, therefore, very different from that which results from disease of the posterior roots, and appears to be a primary degeneration.

There is no reason why a person with amyotrophic lateral sclerosis should not acquire *tabes*, but what is the meaning of an apparently primary degeneration of Goll's

¹³ Hektoen: *Jour. of Nervous and Mental Disease*, 1895, p. 145.

columns such as has been noted in the case of spastic paralysis reported by Dejerine and Sottas (l. c.) or in our case and other cases of amyotrophic lateral sclerosis? In our case no symptoms indicated the existence of this slight sclerosis in the columns of Goll.

The description of the pathological condition in amyotrophic lateral sclerosis as held by Charcot has not been accepted by all. Chief among the opposers Leyden may be mentioned. He has refused to accept amyotrophic lateral sclerosis as a sharply defined disease and believes it cannot be separated from progressive bulbar paralysis. He does not regard the spasticity as of great importance. Gowers also has not accepted Charcot's views. The case reported by Senator¹⁴, and later by Wolff¹⁵, does not favor Charcot's idea that the pyramidal tracts are affected in advance of the motor cells of the cord. This case is well known, and we need not refer to it in detail. The symptoms of amyotrophic lateral sclerosis were present in a person in whom the spinal-motor cells of the cervical and thoracic regions were atrophied, and the pyramidal tracts were not degenerated. However, foci of softening were found in the cord, and the brain was not examined. The muscular atrophy is easily explained by the condition of the motor spinal cells. May we not venture to suggest that the foci of softening had some connection with the spasticity? Senator would have us use the name of bulbo-spinal spastic atrophic paralysis instead of amyotrophic lateral sclerosis.

We know that spasticity may be produced by cerebral lesions without sclerosis of the pyramidal tracts. We, ourselves, have seen cases in which the lesions occurred in early childhood and sclerosis of the motor tracts did not develop. Long ago Schulz¹⁶ reported two cases without alteration of the spinal cord presenting the appearance

¹⁴ Senator: *Deutsche med., Wochenschrift*, vol. xx., 1894, p. 433.

¹⁵ Wolff: *Zeitschrift für klin., Med.*, xxv., p. 326.

¹⁶ Schulz: *Deutsches Archiv., für klin., Med.*, 23, p. 343.

of spastic spinal paralysis (lateral sclerosis). In one a cerebral tumor was found, in the other chronic internal hydrocephalus.

We can easily understand, therefore, that while degeneration of the central and peripheral motor tracts has been found in most cases of amyotrophic lateral sclerosis, lesions of the peripheral motor neurons with some destruction of tissue in the brain or upper part of the cord, affecting the motor tracts indirectly, might also produce the clinical picture of amyotrophic lateral sclerosis, and yet the lateral columns be apparently intact.

The fact that the bulbar nuclei were, for the most part, normal in our case, while the degeneration of the motor fibres extended above these nuclei, seems to support Charcot's view in regard to the primary nature of the degeneration in the motor tracts, for it is in this way we would explain the symptoms of bulbar paralysis.

The case reported by Strümpell¹⁷ would seem to justify Charcot's view, for certain instances at least. The pyramidal tracts were degenerated from the cerebrum to the lower lumbar region, and the cells of the hypoglossal nuclei and those of the anterior horns of the cord were not normal. The changes were much greater in the pyramidal tracts than in the cells, and Strümpell himself believed that the lesions originated in the central motor neurons.

Raymond¹⁸ acknowledges that in the majority of cases amyotrophic lateral sclerosis cannot be separated from the labio-glosso-laryngeal paralysis, but he believes that in some cases the atrophy of the spinal motor cells or of the bulbar nuclei may be without involvement of the pyramidal tracts. He published in collaboration with Mathias Duval¹⁹ a case in which the bulbar nuclei were affected

¹⁷ Strümpell: *Deutsche Zeitschrift für Nervenheilkunde*, vol. v., p. 225.

¹⁸ Raymond: *Leçons sur les Maladies du Système Nerveux*, Première Série.

¹⁹ Duval and Raymond: *Archives de Physiologie*, 1879.

and no trace of sclerosis of the motor tracts was seen. Raymond refers to a number of other cases of pure labio-glosso-laryngeal paralysis,—pure in the sense of being without involvement of the motor tracts.

The arrest of the degeneration in our case in the bulbo-pontine regions follows the usual rule, though in some instances the degeneration has been traced higher, even to the cortex (Kojewnikoff²⁰, Charcot and Marie²¹, Lemmelin²², Lombroso²³, Nonne²⁴, and Mott²⁵).

Charcot and Marie reported two cases in which they found great diminution of the large pyramidal cells normally present in the paracentral lobule. The other cells of the cortex appeared to be normal. We take it that they refer to the giant cells of Betz. These are present in our case, but possibly are not as numerous as usual.

Marie²⁶ called especial attention to the fact that the degeneration of the lateral columns in amyotrophic lateral sclerosis involves a greater area than that occupied by the pyramidal tracts, as marked out by secondary degeneration from cerebral lesions, or by the myelination of the white columns. This degeneration in the lateral columns, extending ventrally beyond the crossed pyramidal tracts in an area called the supplementary zone (Marie, Brissaud), was supposed by Marie to be the result of degeneration of column cells in the spinal cinerea. Brissaud (l. c.) eagerly accepted this teaching and added to it. According to him, the pyramidal fibres in amyotrophic lateral sclerosis are not affected primarily, and even when the process has lasted some years, normal fibres may be found in the sclerotic areas. He believes that the malady involves

²⁰ Kojewnikoff: *Archives de Neurologie*, vol. vi., 1883, p. 356.

²¹ Charcot and Marie: *Archives de Neurologie*, x., 1885, pp. 1 and 168.

²² Lemmelin: *Upsala lakaref*, 1887, xxii., No. 7, p. 299, cited by Raymond.

²³ Lombroso: Cited by Joffroy and Achard.

²⁴ Nonne: *Neurologisches Centralblatt*, 1894, p. 393.

²⁵ Mott: *Brain*, vol. xviii., p. 21.

²⁶ Marie: *Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris*, 1893, p. 757.

primarily only short fibres, and that the pyramidal fibres may be affected secondarily. The short neurons throughout the cerebro-spinal axis are supposed to be diseased. This view does not seem to have gained many adherents.

J. B. Charcot²⁷ speaks especially of the degeneration of this supplementary zone in progressive spinal muscular atrophy and in this disease the pyramidal tracts are not affected. The fact that the pyramidal tracts are not degenerated shows that something more than degeneration of tract cells with their axis cylinders must occur in amyotrophic lateral sclerosis. The degeneration of the supplementary zone does not seem to be present in every case of progressive spinal muscular atrophy. In one of Dejerine's²⁸ two cases it was not detected and in the other it was so slight that its existence was questionable.

It is hardly fair to Oppenheim²⁹ to ignore the statement made by him in 1892 that the degeneration of this supplementary zone is due partially to atrophy of short fibres which degenerate secondarily to disease of the anterior horns. This supplementary zone is largest in the cervical and thoracic regions (Mârie).

The tongue in our case does not show atrophy when examined microscopically. We have employed the method of Marchi in the hope of finding accumulations of fat drops within the muscular fibres, such as Obersteiner³⁰ has described in the tongue from a case of tabes, but we have not been successful. Joffroy and Achard³¹ succeeded in finding atrophy of the lingual muscles in a case of amyotrophic lateral sclerosis, and the atrophy seemed to be more marked near the tip of the tongue.

²⁷ J. B. Charcot, Thèse de Paris, 1895, Review in *Revue Neurologique*, 1895, p. 502.

²⁸ Dejerine: *Comptes rendus de la Société de Biologie*, 1895, p. 188.

²⁹ Oppenheim: *Arch. für Psychiatrie*, xxiv., p. 758.

³⁰ Obersteiner: *Arbeiten aus dem Institut für Anatomie und Physiologie*, etc., iii.

³¹ Joffroy and Achard: *Archives de Médecine Expérimentale et d'Anatomie Pathologique*, 1890, vol. 2, p. 441.

Kronthal³² also has found the lingual muscles degenerated in amyotrophic lateral sclerosis.

The posterior longitudinal bundle has been found degenerated in a number of cases of amyotrophic lateral sclerosis (Muratoff³³, Hoche³⁴, Mott (l. c.), and it is believed that the fibres of this bundle unite the nuclei of the motor cranial nerves. Muratoff (l. c.) found this bundle abnormal in three cases in which the nuclei of the motor cranial nerves were altered and normal in a fourth case in which these nuclei were not affected. This posterior longitudinal bundle is analogous to the anterolateral ground bundle of the cord, and Muratoff believes that he was the first to find the former degenerated in amyotrophic lateral sclerosis. Muratoff, writing in 1891, states that the anterolateral ground bundle had been found degenerated in amyotrophic lateral sclerosis by Moeli (poliomyelitis), Flechsig, Leyden, Vierordt, Gombault, Charcot and Marie, Roth, Strümpell and himself. Since this date many other observers have remarked upon this degeneration.

Oppenheim mentions especially that he found the posterior longitudinal bundle normal, contrary to the findings of Muratoff.

In our case this bundle of fibres is not degenerated, and its integrity is probably due to the fact that most of the motor cranial nuclei are apparently normal.

The hypoglossus nucleus and nucleus ambiguus in our case do not seem to be much affected, and the intrabulbar portions of the nerves arising in these nuclei are not degenerated. The cells of the posterior nucleus of the vagus are greatly degenerated and many of them consist almost entirely of so-called pigment. This may at first thought cause some surprise, for the nucleus is

³² Kronthal: *Neurologisches Centralblatt*, 1891, p. 133.

³³ Muratoff: *Neurologisches Centralblatt*, 1891, p. 512.

³⁴ Hoche: *Neurologisches Centralblatt*, 1897, p. 242.

supposed to be sensory. Degeneration of these cells has been observed in a number of cases of amyotrophic lateral sclerosis. Muratoff in three cases of this disease, found atrophy of the hypoglossus nucleus, complete atrophy of the roots of the twelfth nerve in two cases and partial atrophy in a third. In all three cases the posterior nucleus of the tenth nerve was affected, but less so than the

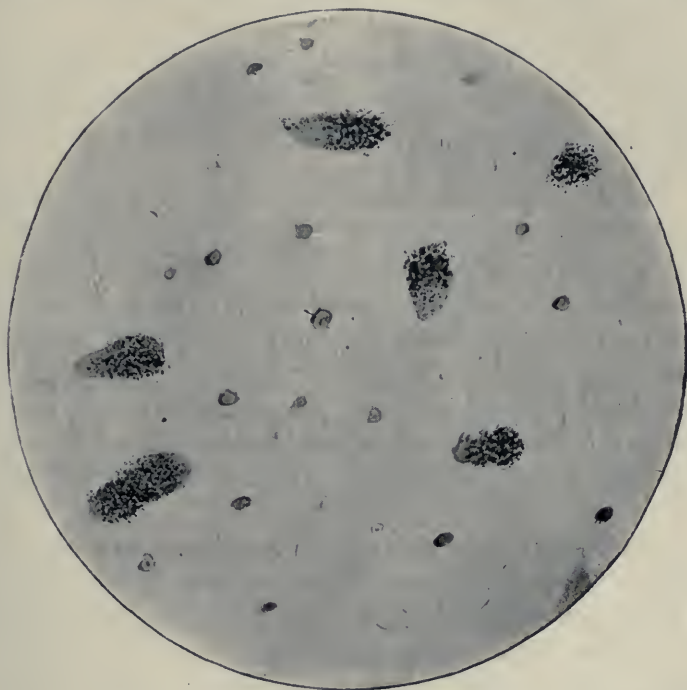


FIG. IV. Pigmentary degeneration of the posterior nucleus of the vagus.

twelfth nucleus, and the nucleus ambiguus was perfectly normal. Oppenheim³⁵ speaks of the difficulty in judging of the condition of the anterior nucleus of the vagus, as its cells do not always form a compact group. In speaking of amyotrophic lateral sclerosis he says that Eisenlohr found the posterior nucleus of the vagus diseased in its

³⁵ Oppenheim: *Archiv. für Psychiatrie*, xxiv., p. 758.

lowest portion. Dejerine also found this posterior nucleus altered, while the anterior was normal, and Freund made a similar observation. Kronthal and he (Oppenheim) each found the posterior nucleus degenerated.

Marinesco³⁶ observed degeneration of the posterior vagus nucleus and concluded that this nucleus must be motor. Van Gehuchten³⁷, however, has offered a different explanation for Marinesco's findings.³⁸

We mention, without attempting to draw any conclusions, that degeneration was found, not only in the posterior nucleus of the vagus, but also in one of the laryngeal muscles examined.

The anterior spinal roots in our case are only slightly degenerated. This is scarcely what we should expect in view of the extensive atrophy of the cells of the anterior horns, but it is well to remember that the condition of these roots is not entirely dependent on that of the cells. Senator³⁹ states that in his case with the symptoms of amyotrophic lateral sclerosis, the anterior spinal roots, even in the cervical and thoracic regions where the motor cells were greatly atrophied, were not notably degenerated.

While the nerves arising in the degenerated cranial nuclei in Kronthal's case of amyotrophic lateral sclerosis were also degenerated, the nerves arising in the cord were normal, although the cells of the anterior horns were entirely degenerated and the anterior roots were much altered. Kronthal states that the peripheral nerves have not often been studied in amyotrophic lateral sclerosis. As the peripheral ramifications of otherwise normal nerves in some cases of amyotrophic lateral sclerosis were

³⁶ Marinesco: *Comptes rendus de la Société de Biologie*, 1897.

³⁷ Van Gehuchten: *Centralblatt für Nervenheilkunde und Psychiatrie*, Beiheft, Oct., 1897, p. 15.

³⁸ Considerable evidence has been offered by Mahaim in regard to the motor functions of the posterior nucleus of the vagus. *Journal de Neurologie*, No. 13, 1898, p. 259.

³⁹ Senator: *Deutsche med. Wochenschrift*, vol. 20, p. 433.

diseased, Kronthal is inclined to believe that the degeneration of muscular tissue affects the nerve terminations. The case of muscular dystrophy studied by one of us (Spiller⁴⁰) demonstrates the fact that the muscles may degenerate completely and the intramuscular nerve bundles still appear perfectly normal. Kronthal⁴¹ is positive that degeneration of the peripheral nerves does not necessarily follow degeneration of the cells of the anterior horns of the cord. This cannot be said of the cranial nerves according to his views.

Oppenheim⁴² makes very clear statements in regard to this question. According to him, even when the anterior cells of the cord are almost entirely destroyed, the anterior roots may not be notably altered, and when the anterior cells are normal, or nearly normal, a moderate atrophy of the anterior roots may be found. The explanation for this he is unable to give. Other authors have expressed similar views based on pathological findings.

The nearly normal condition of the motor cranial nerves in our case, notwithstanding the existence of symptoms pointing to bulbar involvement, is not difficult to explain. The pyramidal tract was degenerated above the level of these nuclei and consequently the central neurons of the motor cranial nerves were probably affected, causing in this way symptoms of bulbar paralysis. Oppenheim (l. c.) observed symptoms pointing to disease of the ninth, tenth and eleventh nerves in amyotrophic lateral sclerosis, but no changes in the nuclei of these nerves were found. Hoche⁴³ has been able, in recent hemiplegia and in amyotrophic lateral sclerosis, to trace fibres by the method of Marchi which leave the medial part of the pyramidal tract, pass into the raphe, and cross transversely

⁴⁰ Spiller: *The Journal of Nervous and Mental Disease*, 1897.

⁴¹ Kronthal: *Neurologisches Centralblatt*, 1891, p. 133.

⁴² Oppenheim: *Archiv. für Psychiatrie*, xxiv., p. 758.

⁴³ Hoche: *Neurologisches Centralblatt*, 1897, p. 245, and *Archiv für Psychiatrie*, xxviii., p. 980

to the facial nucleus of the opposite side. He has also traced the connection of the hypoglossus nucleus with the pyramidal tract.

The fibres of the pyramidal tracts are not entirely destroyed in our case of amyotrophic lateral sclerosis, and the degeneration is less intense than that seen after cerebral lesions. This has been noted by several investigators in other cases of this disease.

Hoche⁴⁴ found that the Marchi method did not stain to any extent the pyramidal tracts of the cord, oblongata, and pons in his study of amyotrophic lateral sclerosis, but showed numerous black dots in the crus. The degenerated motor tracts could be traced into the central gyri. This is in favor of the ascending nature of the degeneration, for Marchi's method is only available for recent degeneration, and Hoche's findings are in accordance with the ascending course of the symptoms in many cases. In our case we have sought to find recent degeneration by Marchi's method, but have not been able to employ tissue from a lower level than that of the thalamus. No degeneration of any moment has been detected within the inner capsule, and if a gradually ascending degeneration existed in this case it had not reached the thalamic region.

CONSIDERATIONS ON AMYOTROPHIC LATERAL SCLEROSIS.

BY EDWARD D. FISHER, M.D.

Dr. Fisher considered amyotrophic lateral sclerosis a disease of a character similar to that of progressive muscular atrophy, chronic poliomyelitis, and tabes. All these were simply degenerative diseases of the nervous system. Amyotrophic lateral sclerosis was not an entity, and the name in future would probably be abolished. It was rare, indeed, to find a so-called typical case of progressive mus-

⁴⁴ Hoche: *Neurologisches Centralblatt*, 1897, p. 242.

cular atrophy, in which the lesion was confined to the anterior horns. The same might be said of tabes, the lesion extending beyond the posterior columns, involving the lateral tracts, and also the peripheral nervous system. The so-called systemic or tract diseases were relics of an old-time nomenclature, which would ultimately disappear. In all these diseases the physician had to deal with diffuse affections of the spinal cord, medulla and brain. The author presented microscopical sections from a case which, he said, was a typical clinical picture of amyotrophic lateral sclerosis, and which showed disease of both lateral and posterior columns and of the anterior horns.

Dr. William C. Krauss, of Buffalo, presented some specimens for microscopic examination which showed the characteristic lesions of amyotrophic lateral sclerosis, together with a slight sclerosis of the posterior columns, more particularly of the columns of Goll. He stated that the specimens had been handed to him for examination and he did not know much about the clinical history of the case.

DISCUSSION.

Dr. Dercum referred to the fact that the case reported by himself and Dr. Spiller was in the beginning apparently one of lateral sclerosis, the lateral column symptoms preceding, without any doubt, the muscular atrophy. When the latter did make its appearance, it did so in the usual manner, affecting first the hypothenar and thenar eminences. The bulbar symptoms were probably referable to changes in the cerebral motor tracts above the nuclei of the medulla oblongata.

Previous to his death the man had lost at various times control over the bladder and rectum, and at such times catheterization was necessary; the lesion of the bladder found at the necropsy may have had its origin in this fact. Nevertheless, the lesion certainly indicated an impaired resistance of the tissues of the bladder, and in this sense was trophic.

Dr. Dercum said he was not prepared to go as far as Dr. Fisher in regarding tabes as being actually related to the disease which we know as amyotrophic lateral sclerosis.

Dr. Charles K. Mills, of Philadelphia, thought that the

microscopical studies in a case like the one reported in the paper of Drs. Dercum and Spiller were of great practical value to the clinician. It is only by work of this character that we shall succeed in placing upon a firm foundation the various types of these degenerative nerve diseases, interpret their phenomena, and study them from a more comprehensive standpoint. The literature quoted by Dr. Dercum and Dr. Spiller showed how frequently different portions of the nervous system—different systems of neurons—are attacked in these diseases; it also showed how one is attacked with less intensity than another.

Dr. Mills did not believe that a disease existed which could be properly classed as primary lateral sclerosis. The case reported by Drs. Dercum and Spiller fortified this view, and it was further strengthened by another case of his own which he had not yet reported, but which he had had an opportunity to study for many years and finally to examine on the post-mortem table. When this patient had first come under his observation at the University Hospital it was a question whether the case was one of hysteria of the spastic type, or of primary lateral sclerosis. He remained under more or less constant observation for several years, and during this period the symptoms of so-called primary lateral sclerosis gradually developed, with contractures, and, later, bulbar symptoms.

Dr. Mills expressed the opinion that there are three distinct types, or rather sub-types, of this disease. In one type the symptoms of so-called primary lateral sclerosis develop early and contractures and bulbar symptoms later; in the second type muscular atrophy in the limbs is contemporaneous with the beginning of the spastic condition, and in the third type special bulbar symptoms develop very early.

He was in accord with the remark made by Dr. Fisher that amyotrophic lateral sclerosis should be removed entirely from the classification of spinal diseases. It cannot be regarded as purely cerebral, or spinal, or peripheral, but should be recognized as an affection of a special systems of neurons which have been attacked by a degenerative process, perhaps due to a virus, which may possess an elective affinity for certain neurons.

Dr. P. C. Knapp, of Boston, believed that many neurologists were in accord with Dr. Fisher in his rejection of amyotrophic lateral sclerosis as a pathological entity. The present situation in regard to the classification of these diseases is somewhat analogous to that of the muscular dystrophies. A few years ago several clinical types of the latter were described with great accuracy, according to the groups of muscles af-

fected, and now we have merged those clinical types into one disease.

Dr. Knapp said that while the three types of typical amyotrophic lateral sclerosis, progressive spinal muscular atrophy beginning in the thumb muscles, and typical bulbar paralysis, mentioned by Dr. Mills were well recognized, many transitional types were met with which did not absolutely fit any one of them. We must recognize that there is but one pathological entity in the whole process.

The speaker questioned Dr. Fisher's statement regarding the frequency of syphilis as an etiological factor in these cases. It was certainly much less common in cases of degeneration of the motor region of the central nervous system than of the sensory region, namely, in tabes.

Dr. H. M. Thomas, of Baltimore, said he agreed with Dr. Knapp that no satisfactory distinction could be drawn between the three types of progressive muscular atrophy—amyotrophic lateral sclerosis, progressive muscular atrophy (type Aran-Duchenne) and progressive bulbar paralysis. For the proper understanding of the question we must take into consideration the historical development of this disease. When Charcot described amyotrophic lateral sclerosis all forms of progressive muscular wasting were included in the title, "progressive muscular atrophy," and were believed to be due to a primary disease of the ventral horns. This included what we now call the muscular dystrophies as well as some other diseases. From this great group Charcot distinguished certain cases which had a sharp, clinical picture. The disease began with a spastic paralysis of the arms, and this was followed by a diffuse wasting of the paralyzed muscles. Spastic rigidity of the legs was associated with this, and later the bulbar muscles were involved. The anatomical picture corresponding to this was a lesion in the pyramidal tracts combined with one of the ventral horns. The lesion in the pyramidal tracts, Charcot believed to be primary. To this disease he gave the name amyotrophic lateral sclerosis. About the same time, Leyden described the same anatomical findings in cases of progressive bulbar paralysis. In these cases the spastic condition of the limbs had not been present during life. Leyden thought that the disease might be primarily anywhere in the motor path. After attention had been called to this sclerosis of the pyramidal tracts, it was found more and more frequently; so much so that some authors, Gowers among them, believed that it was a constant lesion and that there were no cases in which the disease was confined to the ventral horns, the lesion which Charcot had considered characteristic of the Aran-

Duchenne type of progressive muscular atrophy. Later observations have shown that certain rare cases occur in which the disease is confined entirely to the lower motor segment. It is, however, impossible to distinguish these cases clinically. The muscular spasm, upon which Charcot laid such stress, occurs, as he described it, in only a few of the cases in which the characteristic lesions of amyotrophic lateral sclerosis are found after death. It may vary in every degree, even to complete flaccidity with loss of the deep reflexes. If the term, amyotrophic lateral sclerosis, is used in its purely anatomical sense, or if it is used to characterize those cases which follow the clinical picture which Charcot drew, it may, perhaps, be allowable, but it cannot be used, as is the custom, in both these senses. The name itself is a bad one, combining as it does a symptom—muscular atrophy—and a disease process of the central nervous system—lateral sclerosis—which are really not dependent upon each other. It seemed to Dr. Thomas better not to use the name and to class these three diseases under the general title of progressive (central) muscular atrophy, for it is probable that they all are simply variations of one and the same disease which sometimes begins in the upper motor segment, sometimes in the lower, and usually involves both. Bulbar paralysis would represent the group in which the motor nuclei in the medulla oblongata are involved early or extensively.

Dr. Spiller, in closing, said he felt perfectly sure that disease of the anterior horns could exist without degeneration of the pyramidal tracts. He stated, in regard to the relation of tabes to amyotrophic lateral sclerosis, that a man might have degeneration of the lateral columns and later a similar affection of the posterior columns; indeed, one process might possibly predispose to the other, but it could not be said that the two diseases were related. Dr. Spiller said that we have no satisfactory evidence that amyotrophic lateral sclerosis is due to a virus.

Dr. Fisher, in closing, said the discussion on the two papers had clearly shown just what the present opinion of neurologists is regarding this class of affections. Eventually, he thought, the influence of the past would be shaken off, and a new and more correct classification of these diseases would result. The pure types of this class of affections, which were formerly described, are as a matter of fact rarely met with; there are many transitional types, but no distinct clinical entities.

REPORT OF A CASE OF TABES WITH HEPATIC CRISES;
AUTOPSY.*

By WILLIAM C. KRAUSS, M.D.,

BUFFALO, N. Y.

In a personal experience with twenty-two cases of tabes dorsalis, I have met with all the major symptoms and nearly all the minor ones of this disease, as depicted in the treatises on nervous diseases and in the special monographs on this subject. In only one case have I encountered phenomena not elsewhere observed by me, and as far as I am able to learn, not heretofore described. It may be presumptuous to call the attacks noted,—crises, or to regard them as part and parcel of the tabetic syndrome, but they resembled closely other commonly observed crises in character and sequence, that I unhesitatingly connect them with the disease, and will characterize them as hepatic crises.

The following peculiar and distressing attacks have been differentiated by various observers, and characterized by Charcot as crises tabétiques; they have been called respectively, according to the organ or part affected, gastric, laryngeal, pharyngeal, bronchial, cardiac, nephralgic, intestinal, vesical, rectal, clitoric or urethral crises. These all agree in so far that they are periodic, paroxysmal, limited to the one organ, are accompanied by pain and motor activity, and call forth the special function of the organ involved. Thus, for instance, the rectal crises are attacks of intense pain in the rectum with tenesmus, and accompanied by a sensation as if a foreign body were to be expelled. Hence in hepatic crises one would expect to find periodic paroxysms of pain over the hepatic area, with jaundice and clay-colored stools not referable to any diseased condition of the liver itself. Such, in truth, were the symptoms observed in the following interesting case:

Mrs. G.; age, 44 years; height, 5 feet 3 inches; weight, 138 pounds; complexion dark.

Family history was negative.

Early history: From childhood to commencement of present trouble she had always enjoyed good health. She gave no history of syphilis, either hereditary or acquired. She menstruated when 14 years old, and married when 23. About eight years before her death she began to have shooting pains in her legs, occurring at intervals and disappearing. Gradually difficulty in locomotion developed, so that she was

*Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

obliged to walk with a cane, then with one crutch, then two crutches, and on this account she sought admission into the St. Francis' Hospital for the Chronic Sick and Aged in 1890. About this time she began to have excruciating pains over the right side of the chest, close to the lower border of the ribs, accompanied by a pronounced jaundice. These spells occurred every four to five weeks, obliging her to keep her bed for two to three days at a time, and then would pass off slowly, leaving her weak and exhausted for several days. The pain was diffuse, neuralgic in character, uninterrupted, deep-seated and very intense, necessitating the administration of large doses of morphia. The jaundice would remain for from four to six days, then would totally disappear. These spells were not accompanied by any nausea, vomiting or gastric disturbance of any kind. She usually partook of fluid food at these times without experiencing any distress; her bowels were regular, stools were clay-colored during these crises; at other times they were normal, and the urine showed traces of bile pigment, but no sugar or albumin.

An examination of her condition in 1890 revealed the following symptoms: Mind was active and acute, memory was good, and she slept well. Her head was not painful to percussion, and she gave no history of headaches.

Eyes: Pupils were contracted; Argyll-Robertson pupil was present. No history of ptosis, double vision, or dimness of vision could be obtained and the ophthalmoscopic examination showed no atrophy of the optic nerve.

No disturbances of the senses of smell, taste or hearing were discoverable.

Strength of the hands was well preserved. No marked inco-ordination of the movements of the hands or arms was seen, and she was able to do some sewing and to make button-holes for coatmakers. The legs, on the contrary, showed an extreme degree of inco-ordination, the gait was markedly ataxic, Romberg's symptom was present and the tendon reflexes were abolished. No disturbances of sensation existed, and the vesical and rectal reflexes were also undisturbed.

Palpation of the abdomen revealed no hepatic enlargement, no tenderness over the hepatic area, or over the abdomen. This was in marked contrast to the condition present during one of the crises. The hepatic area was then so sensitive that she could not bear the weight of her clothing, and she immediately went to bed when the attack was about to begin.

Examination of the feces showed no presence of bile, and no gall stones. The urine was highly colored, and gave the bile reactions.

During these attacks, the skin, mucous membranes, conjunctivæ, and even the scalp were all highly jaundiced, and she complained of the bitter taste in her food.

The attack lasted two or three days, and the intense pain was partially relieved by large doses of morphia hypodermically administered. Her condition following one of these attacks was one of great debility and prostration for fully a week. She then slowly recovered and was able to sew and take her daily suspensions.

The ataxia became so pronounced that she was unable to walk from her bed to the chair, and eventually had to be carried, until she became totally bedridden.

She gradually lost in strength; the hepatic disturbance became more and more intense, and in April, 1894, she died from inanition.

The autopsy was made the day after her death; special attention being directed to the brain, spinal cord and liver.

The brain was apparently unaffected; the dura and pia were moderately injected, were not adherent, and the Pacchionian bodies were not enlarged.

The spinal cord was not abnormal to the eye, and after the removal of the dura, the cord was placed in Müller's fluid for microscopical examination.

The heart and lungs, likewise the abdominal viscera, were normal in size, appearance and consistency. The liver was not enlarged, and weighed $3\frac{1}{4}$ pounds. Its capsule was not adherent, and on section was firm and uniform throughout. The hepatic vessels were moderately engorged with blood. The cut surface showed slightly the mottled appearance characteristic of nutmeg liver, and the hepatic and portal vessels contained a large quantity of blood.

The gall bladder was filled with bile, contained no gall stones, and the ductus choledochus and accessory ducts were passable with a sound.

The liver was cut into small cubes about one inch thick, so that no new growth of any kind would be overlooked, and many of these cubes were placed in alcohol and Müller's fluid for further examination.

After the usual hardening and imbedding methods had been employed, the liver and spinal cord were cut and stained with the carmine, hematoxylin, and by Weigert's method, and mounted in balsam.

The spinal cord sections showed sclerosis of the posterior tracts throughout the entire length of the cord. The peripheral nerves were not examined.

The liver appeared to be in the early stages of chronic congestion, otherwise known as nutmeg liver. There was,

however, little atrophy of the cells about the central vein, and but very little pigment scattered throughout the cells. The vessels were not particularly engorged, nor had any hypertrophy taken place in their walls.

As a result of the microscopical examination, we find the characteristic lesions in the posterior tracts of the cord, and a nutmeg liver in its early or imperfect state.

The latter was without any cardiac or pulmonary disease, or any new growth producing pressure on the inferior vena cava or obstructing the return of venous blood to the thoracic organs. Neither were any of the sequelæ or concomitants of chronic venous engorgement present; such as ascites, hemorrhoids, varices, intestinal catarrh or diarrhea.

No condition was present, therefore, which would explain the mechanism of this congestion, which was purely a mechanical one. On the other hand, no condition was present in the liver, to explain the symptoms presented by this patient. No neoplasm of any kind, no hepatitis, and no gall stones were found. A chronic congested liver is not accompanied by any pain whatever, does not produce a very high degree of jaundice, does not cause paroxysmal symptoms, and can often be diagnosed by its enlargement.

It would seem that some disturbance to the nerve supply of the liver and gall bladder was present in this case, possibly producing constriction of the bile ducts and a consequent damming back of the bile secretion.

The occurrence of crises in *tabes dorsalis* is rather rare, as in the twenty-two cases observed by me, only one other case presented these symptoms, and they were referable to the stomach; were gastric crises. Riley¹ in his study of sixty-one cases of *tabes* found gastric crises in eleven cases and laryngeal crises in six cases. No other crises were observed. Olivet,² in studying the cases of *tabes* at the Göttingen clinic from 1877 to 1889, forty-three cases in all, found only four patients suffering from gastric crises. In the four hundred cases studied by Leimbach³ in Professor Erb's clinic, only six per cent. were found suffering with tabetic crises.

As a rule the crises affect only one organ, but Grabower⁴ reported a case to the Berlin Neurological Society in 1896, in which laryngeal, pharyngeal and gastric crises were present in the same patient.

¹Journal of Nervous and Mental Disease, Sept., 1898.

²Inaugural Dissertation, Univ. Göttingen, 1891.

³Zeitschrift f. Nervenheilkunde, 1895, VII.

⁴Neurologisches Centralblatt, Feb. 1. 1896.

DISCUSSION.

Dr. William Osler inquired whether any fever accompanied the patient's attacks of liver trouble, and how long before her death they had ceased. The pain and jaundice accompanying the attacks would lead one to suspect the presence of gall stones. Even though no gall stones were found at the necropsy that did not exclude the possibility that they were present during life and gave rise to the hepatic symptoms.

Dr. Krauss, in answer to Dr. Osler, said the patient had had no fever whatever during her hepatic crises, and she had had these attacks until the time of her death. In fact, she was in a jaundiced condition when she died. The fæces were examined almost every day, but no traces of gall stones were ever found.

MINUTE ON THE DEATH OF DR. E. C. SEGUIN.

The committee appointed by the American Neurological Association to take action regarding the death of the late Dr. Edward Constant Seguin report the following minute:

The association has learned with deep sorrow of the death of Dr. Seguin. He was one of the charter members of the association; for many years he acted most efficiently as its secretary and treasurer; and he was its president at the fifteenth annual meeting held in 1889.

Some of his most valuable contributions to neurological medicine were in the form of papers read before this association, and he frequently took part in the debates of the association, his remarks always being well considered and of permanent value. The older members of the association will recall the continuous and close interest which he took in its proceedings. He was courteous in his bearing, but was courageous in the assertion of his opinions and in the maintenance of his positions. He was always appreciative of the merits of his colleagues.

As a teacher and writer his work is of the very highest order. As a neurological practitioner he has probably never been excelled. No physician ever gave closer or more painstaking attention to those by whom he was consulted.

In common with his father he took great interest in the study both of idiocy and of medical thermometry, adding much of great value to our knowledge of these subjects. He was largely instrumental in introducing to the profession in this country the use both of general and surface thermometry.

Dr. Seguin's contributions to neurology and to general medicine have given him a prominent position in medical

literature. He and one or two others may be regarded as the pioneers of American neurology. His contributions to the therapeutics of nervous diseases are especially valuable. His work entitled "Opera Minora" contains a number of papers of great scientific and practical value. His pathological investigations into the nervous system were among the most important in the history of American neurology in the first ten years of the existence of this association.

J. J. PUTNAM,

L. C. GRAY,

C. K. MILLS.

THE FORMATION AND EXCRETION OF THE METAPLASM GRANULES OF THE NEURON.*

BY IRA VAN GIESON, M.D.

Dr. Van Gieson described the constituents of the ganglion cells and referred to the reticulum, which transmits the impulses and subserves the phenomena of retraction and extension of the neuron. The speaker said that when the food supply of the ganglion cell is diminished—as it is in alcoholism, in over-fatigue, etc.—we get an indication of it in an excretion of particles which escape from the cell, pass out of the lymph space and cluster about the blood vessels. These metaplastic elements are the results of over-fatigue—of a diminished food supply to the nerve cells. Dr. Van Gieson said that the chemic processes which accompanied this destructive metaplasia of ganglion cells must still be worked out. He had come to realize the fact that these nerve changes were not altogether of toxic origin, but that we must also take into consideration the quantitative and qualitative food supply of the cells.

Dr. Van Gieson thought that in amaurotic family idiocy there was an indication of a diminished food supply to the nerve cells. He agreed with Dr. Sachs that it was bad to make a distinction between arrested development and pathological processes. If we find a degenerative process in an adult, we call it by its proper name; in a child, we call it arrested development. One is the result of the other; degeneration is the result of arrested development. He suggested the name of chronic parenchymatous degeneration in infancy.

*Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

DISCUSSION.

Dr. Dercum inquired whether the process described by Dr. Van Gieson—the excretion by the nerve cell of these metaplasmin granules—was not in a measure a normal process?

Dr. Sachs asked Dr. Van Gieson whether he thought it conceivable that the metaplastic changes he had described were due to a condition of starvation. If so, the question might arise whether the cell changes described in amaurotic family idiocy were primary or secondary, for children afflicted with that disease die in a state of marasmus.

Dr. J. J. Putnam thought that the explanation given by Dr. Van Gieson of the metaplastic changes in the nerve cells, as a result of interference with their nutrition, indicated that a much greater interdependence exists between the nerve tissues and other parts of the body than we now fully recognize. That such a thing is possible, and even probable, is evidenced by what we know of other conditions; for example, the influence exerted by the thyroid on the general nutrition of the body.

A CASE OF ERYTHROMELALGIA WITH MICROSCOPICAL
EXAMINATION OF TISSUE FROM AN AMPUTATED
TOE.*

BY S. WEIR MITCHELL, M.D., LL.D., AND WM. G. SPILLER, M.D.

Drs. Mitchell and Spiller reported a case of erythromelalgia in which the signs of the disease were almost confined to one of the great toes. Amputation of the toe had been resorted to, although considerable doubt was felt as to the benefit to be derived from the operation. The nerves of the toe were intensely degenerated, and the vessels presented a high degree of arteriosclerosis. The amputated bones were larger than the corresponding ones in a normal adult male. The case reported by Auerbach and the one by Mitchell and Spiller are the only cases on record in which important pathological changes have been found, since the disease was first described in 1872. Drs. Mitchell and Spiller believed that involvement of the sensory fibres anywhere between the spinal cord—or possibly within the spinal cord—and the peripheral ramifications is capable, under certain circumstances, of causing erythromelalgia; though hysteria may present similar symptoms.

*Read at the twenty-four annual meeting of the American Neurological Association, May, 1898.

A CASE OF SYRINGOMYELIA WITH UNUSUAL SYMPTOMS; AUTOPSY; MICROSCOPICAL REPORT.*

BY W. N. BULLARD, M.D. AND J. J. THOMAS, M.D.

Drs. Bullard and Thomas reported the case of a boy, previously healthy and of fairly healthy antecedents, who, when three years old, became subject to attacks of headache of much severity, lasting an hour or more; these increased in frequency and severity, and were soon accompanied by vomiting. This condition continued three years, growing gradually worse, and at the end of that time staggering was noticed and a severe double optic neuritis occurred, which soon produced double optic atrophy and nearly total blindness. The headaches, vomiting attacks and blindness continued much the same for nearly two years—the remainder of the patient's life. At times the pain and prostration were such that he was in bed for days or weeks at a time; then he would improve, and would be able to be up and about like a healthy child. During this time the general adipose tissue increased greatly, so that the patient became obese; and to this condition were added, later, exophthalmos and an appearance suggesting myxedema. These two symptoms, however, disappeared after a month or two. During the first part of this time (in March, 1895) temporary paresis of both external recti and nystagmus were noticed. No further changes in the symptoms occurred until about ten months after the blindness began, when paraplegia, accompanied by incontinence of urine and feces, began suddenly and became permanent. At the same time a temporary paralysis of the left face and some weakness of the upper extremities appeared. Bed-sores developed, but later improved, and the child was in good general condition, although the obesity had somewhat diminished at the time of death, which resulted from typhoid fever.

At the autopsy a marked dilatation of all the ventricles and of the iter was found. The branching cavity in the cord seemed to originate in the gray matter, and not to be a diverticulum of the central canal, although at the point of greatest dilatation of the cavity, in the lower cervical region, the central canal opened into and became a part of the general cavity. Above the point of greatest dilatation of the cavity was an ascending degeneration of the direct cerebellar tract, the tract of Gowers, and the posterior columns. Below this point marked degeneration of the direct and crossed pyramidal tracts, diffuse atrophy of the posterior columns, and well-marked

*Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

atrophy of the tract of Gowers were found. At the point of greatest dilatation of the cavity were extensive degeneration of all the fibres of the white matter of the cord, and marked distortion of the gray matter, in which, however, fairly normal nerve cells in moderate numbers were found. The neuroglia fibres in the atrophied tracts were increased, and the neuroglia proliferation about the cavity in the cord was distinct.

THE NEUROLOGICAL ASPECT OF PUBLIC-SCHOOL EDUCATION.*

BY JOHN PUNTON, M.D.

Dr. Punton believed that serious defects still existed in the routine methods of public-school education and training of the young, which were attested not only by the general public but also by the teachers and officers of schools. Moreover, all recognized that marked constitutional differences and defects existed in both mind and body in children of every class of society, but the special relation which they bore to modern educational methods was not so well understood as it should be. The vital relation of these lesions of organization to the proper protection and health of the race on the one hand, and the prevailing vices and social evils on the other, together with their special relation to public-school education, presented a phase of the subject which so far had received but little attention in America, but which really constituted the neurological aspect of education. What was necessary to be done, nay, even demanded, was to fix certain definite standards of weight and measurements for every age and height; any pupil found to be above or below this standard should be treated accordingly. In almost every city in the world anthropometrical investigations had now been made, and the unanimous verdict had been reached that the mental output of the pupil was directly related to his height, weight and physical measurements, and that there was a physical basis for precocity on the one hand and mental dullness on the other.

DISCUSSION.

Dr. Worcester expressed his entire approval of the contents of this paper, and his belief that the present system of education for young children, at least, is radically defective. He believed that children were kept steadily in school at work which is often distasteful to them, until they got a strong dislike for study, and that the results, in the matter of acquisition

*Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

even, are not as good as they might be by other methods. He had in mind a boy who went to school in a very desultory fashion until fourteen years of age, and was then sent to the city high school. He could not have passed the examination for admission to that grade, but he was allowed to go in as an outsider; yet from the outset he stood among the first in his class, and kept his position until graduation. There were probably other boys just as bright who had been going to school every day since they had arrived at school age, and were not in advance of the boy who had "run wild" until the age of fourteen.

Dr. Punton remarked that his object in writing the paper was to direct the attention of the members of the association to this subject.

21. UEBER DIE CHIRURGISCHE BEHANDLUNG DER HIRNSYPHILIS (Concerning the Surgical Treatment of Cerebral Syphilis). F. v. Friedländer and H. Schlesinger (Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, Vol. III, No. 2).

A case is reported in which a gumma was removed from the lower part of the central gyri. An almost isolated cortical hypoglossus paralysis is mentioned, among other interesting features. This is an evidence of the existence of a distinct cortical hypoglossus center in man, similar to that shown by Beevor and Horsley in the orang-outang, and probably located in the lowest fourth of the anterior central gyrus. Vasomotor disturbance was noted on the paralyzed side except in the face, and this, the writer believes, shows the existence of a cortical vasomotor center in man. Pronounced hyperalgesia, delayed and repetition of sensation were observed on the paralyzed side for about three weeks after the operation. Attacks of pain and hyperalgesia in the affected limbs in cases of tumor in the central gyri have been noted repeatedly, but delayed sensation of pain has, heretofore, been found only in spinal and peripheral nerve lesions. This case, therefore, shows that delayed sensation of pain may be due to cortical lesions. This phenomenon is explained as summation of irritation. The case seems to indicate also that the temperature sense has a cortical representation not identical with that of the other sensations.

The authors believe that in cases in which a distinct history of syphilis has been obtained, and in which antisyphilitic treatment has failed, or the tumor threatens life, surgical interference is proper, provided the symptoms do not indicate a tumor of very large size. The indications for operation are:

1. Symptoms indicating a tumor of slow development, accessible and small, when an antisyphilitic treatment has failed.
2. Progression of the symptoms in spite of antisyphilitic treatment, when danger to life exists.
3. Jacksonian epilepsy after antisyphilitic treatment has been employed, and after the other symptoms of tumor have disappeared.

The contraindications are:

1. Symptoms of basal or extensive spinal syphilis.
2. Great loss of strength, amyloid degeneration and other complications of vital organs.

SPILLER.

Society Reports.

THE NEW YORK NEUROLOGICAL SOCIETY.

December 6th, 1898.

The President, Dr. Frederick Peterson, in the Chair.

A CASE OF ACROMEGALY.

Dr. W. M. Leszynsky presented a man, thirty-six years of age, who had been a steady drinker of whisky. About one year ago his sight became blurred. Numbness had been felt in the arms and hands during the past year. About five years ago the patient first noticed that the hands and feet had grown large. They have not grown much during the last two years. Examination showed him to be six feet one inch in height and to weigh 245 pounds. The lower jaw projected half an inch beyond the upper; the lower lip was thickened; the tongue was two inches and a half wide and its surface was cracked. The bones of the face were generally enlarged, the nose being broadened and lengthened and the nostrils widened. The thyroid gland was not enlarged. His pulse was 84. The viscera were normal. The bones of both the upper and lower extremities were all enlarged. There was no atrophy, tremor, ataxia or objective sensory disturbance. The triceps reflex and the gait were normal. From the heel to the tip of the toes measured $11\frac{1}{4}$ inches. The tendo-Achillis and plantar reflexes were feeble, and the plantar reflex was absent; the patellar reflexes were normal. The urine was negative. The case being one of chronic alcoholism at the beginning, the question arose as to whether the eye symptoms were due to alcoholism or to enlargement of the hypophysis. Dr. Leszynsky said that he believed they were due to disease of the hypophysis and to pressure on the optic tract. The reaction of the pupils to light was very sluggish. The vision of both eyes was 20-15. There was no stereoscopic vision; accommodation was normal, and there was no central color scotoma. The field for form and white in the right eye was entirely normal, while the field for green was greatly contracted. Almost complete temporal hemianopsia for white and form was found in the left eye, while the fields for green, blue and red were contracted. The ophthalmoscope showed both optic papillæ pale and atrophic. The sense of smell was entirely absent.

Dr. M. Allen Starr said that he had had under observation a patient with acromegaly, and had given him thyroid extract pretty regularly for four or five years. While taking it he was free from

distressing headache. A slight reduction had occurred in the circumference of the fingers and feet, but this was so slight that it was probably due to the decrease in the general deposit of adipose.

Dr. B. Sachs said that he had observed a case of acromegaly during a period of ten years, and that all sorts of medicinal treatment had been tried without avail.

Dr. Leszynsky thought that the peculiar eye symptoms, the fact that there was no central color scotoma, and that the field for white in one eye was perfect, while those for colors were completely obliterated, helped one materially in making the diagnosis; so that even without the acromegaly one might expect the existence of a growth in the hypophysis. The case was interesting because of the possibility of making an early diagnosis.

A PECULIAR PHASE OF LOCOMOTOR ATAXIA.

Dr. Sachs, on behalf of Dr. Freeman F. Ward, presented a patient showing a peculiar phase of locomotor ataxia. The patient, who had double optic nerve atrophy, was presented for inspection. He showed the Romberg symptom very markedly, falling over immediately on closing the eyes. He was fifty-three years of age, and stated that his present trouble began fourteen years ago with gradual failure of vision.

Dr. Leszynsky recalled having seen a woman with tabes, who was unable to perceive light because of optic atrophy, yet who exhibited a considerable ataxia when the feet were placed together, but in her case the moment she closed her eyes the ataxia disappeared.

Dr. H. M. Thomas, of Baltimore, said that in his list of tabetic cases he had come across the Romberg symptom twice in those who were blind. He had also seen it mentioned several times in literature.

LESION OF THE CHIASM.

Dr. Philip Meirowitz presented a married man, twenty-eight years of age, who had lost completely the sight in the right eye, and in whom the vision in the other eye was very much impaired. The impairment of vision began two years ago, shortly after the patient witnessed a fatal accident to a child. Two days after this accident the patient had anorexia and insomnia, and experienced difficulty in banishing the incident from his mind. Two or three months later he began to be troubled with vertigo, except while in the dorsal decubitus. These attacks of vertigo recurred monthly for five or six months, and then ceased. About the time of the first attack, in May, 1897, the vision of the right eye became impaired, and a few weeks ago this eye became blind. Gradually the temporal half of the left visual field became smaller, until there was almost complete hemianopsia. He had never had severe headaches or pains or motor disturbances. His speech was normal. When examined on November 8, 1898, a slight weakness of the external rectus was noted; the pupils were irregular and larger

than normal; and on looking to the right there was slight nystagmus. The ophthalmoscope showed descending atrophy. Vision in the right eye was zero, and in the left 17-50. There was no history of alcoholism or syphilis. The case was presented as a lesion of the chiasm, and to elicit discussion on the possible value of surgical treatment.

Dr. Graeme M. Hammond said that he would consider the case one of a gradually progressive lesion affecting the right optic nerve at its exit from the commissure. The probability was that the other half of the left retina would be lost. A surgical operation for the removal of a possible neoplasm must necessarily be a severe one, but a surgeon who had experimented on the cadaver with this in view had confidently asserted that a surgical procedure was feasible.

Dr. Leszynsky had seen this man and had carefully studied his case. He had also observed a number of similar cases at one time in conjunction with Dr. Seguin. He was of the opinion that the case would go on to complete atrophy and blindness, and he did not think any operation was justifiable, as he could see no trustworthy evidence of tumor. The patients observed by Dr. Seguin and others had lived for twenty-five or thirty years, although blind.

Dr. Starr said that the case closely corresponded to one that he had had under observation for nine years. This patient had consulted Sir William Gowers, of London, on two or three occasions, and had also seen Horsley, who had pronounced it perfectly impossible to operate upon a tumor in or about the optic chiasm. Gowers had insisted, however, that the case was not one of tumor, but of optic atrophy, and this view had been borne out by the fact that the man, while totally blind, was still alive and fairly comfortable.

Dr. Peterson had seen the case just presented, and had come to the conclusion that it was one of those of atrophy in which the progress is irregular and unusual. A year or two ago he had shown a case of tabes in which hemianopsia had been found at first and finally the loss of three quadrants in the two eyes occurred, undoubtedly as a result of optic atrophy. Without any evidence of tumor the diagnosis of neoplasm did not seem to him warrantable.

STATISTICAL STUDY OF CASES OF TABES EXAMINED AT THE JOHNS HOPKINS HOSPITAL.

Dr. H. M. Thomas, of Baltimore, read this paper, which was founded on 111 cases of tabes presenting more or less complete histories. Of this number, 106 were white persons and five negroes. The negroes represented a little more than 10 per cent. of the cases treated, so that the percentage of tabetics among the negroes was less than half what it should be if the negroes were represented in proper proportion. Syphilis is considered by the majority of physicians to be the cause of tabes. The records show that while the negroes represented only 6.39 per cent. of the total number of males treated in a given time, they constituted 27.63 per cent. of the whole number of males treated for syphilis. In the dispensary the percentage of women suffering from tabes was small—a little

more than 9 per cent. Five female tabetics from the higher strata of society, which was contrary to the usual belief, were seen in the private wards. He had as yet seen no case of tabes in a negro woman. His statistics presented nothing of much interest regarding the age at which the disease develops. Most of the cases developed between thirty and fifty years, the youngest being twenty-five and the oldest sixty-six years. The duration of the disease could be determined with fair accuracy in 107 cases. In eleven it lasted ten years or more; in one the duration was thirty years. He had definite notes in ninety-five of the ninety-seven male cases. The percentage of cases in which syphilitic infection was certain was forty-two; the percentage of possible syphilitic cases was sixty-three. The author's conclusions regarding the relation of syphilis to tabes are: (1) In a large proportion of cases of tabes a history of syphilis can be obtained; (2) in a certain not inconsiderable number there is no history of venereal sore or other syphilitic manifestation; (3) in negroes tabes is relatively uncommon, whereas syphilis is very much more common in them than in white persons; (4) the partial immunity of women is greater than can be satisfactorily accounted for by the relative infrequency of syphilis among them.

^ This study seemed to show that syphilis is not the only factor in the development of tabes. The time elapsing between the syphilitic infection and the first symptom of tabes was determined in forty-seven cases. The shortest interval was two years, and the longer ones twenty-six, twenty-seven, thirty, and a doubtful forty-two years. Pain occurred in fifty-seven as the initial symptom; ataxia in twenty-four; numbness of the extremities in six; paralysis of the bladder in five; nausea and vomiting and gastric crises in four. One patient complained that at first he had suffered pain extending from the penis to the rectum, and since this paper had been written he had seen another such case. Optic atrophy occurred in eleven; eye-muscle paralysis in thirty-three; Argyll-Robertson pupil in seventy; both pupils immovable to light but reacting to accommodation in fifty-seven cases. Of the cases showing optic atrophy, the ataxia was marked in two; slight in eight; absent in one. In two of the cases the Romberg symptom was marked in spite of blindness. In seventy-five cases in which the sexual power was noted, both power and desire were lost in thirty-eight, and in three of these was preceded by a marked increase; it was weakened in twenty-four; in one the power was lost, but the desire retained; in one both were increased; in ten both were normal. The muscular sense was disturbed in thirty-eight out of forty-four cases. Charcot's joints occurred typically in five, and there was suspicious enlargement in three

others. In seven cases showing mental symptoms general paresis was suggested.

In conclusion, Dr. Thomas said that too much weight should not be given such a statistical study as this, and it should not be used as a basis for too definite statements as to the relative frequency of any particular symptom.

Dr. Starr said that the statistics of one clinic should be studied in conjunction with those from other clinics. He had had the cases at the Vanderbilt Clinic tabulated up to March 15, 1897. To these 175 cases he had added 98 from private practice, making a total of 273 cases. The males were practically ten times more numerous than the females. The great preponderance of cases developed between the ages of thirty and forty, though 21 were over fifty, and 2 were under twenty. Syphilis was certainly present in 50 per cent., and if these were grouped with those in which syphilis was probable or doubtful, the total would represent 72 per cent. Of the 273 cases, 27 had gastric crises; 21 had optic atrophy; 5 had Charcot's joints; 5 had ulcer of the foot. The most frequent symptom was the pain; next the ataxia; thirdly the bladder disturbance; fourthly numbness of the legs; fifthly girdle sensation, and sixthly ocular paralysis. The knee-jerks were absent in 246 cases; the Argyll-Robertson pupil was present in 184; the Romberg symptom was present in 229. Rectal crises, or severe pains in the perineum, were noted in one case.

Dr. Sachs said that he had always maintained that statistics do not amount to much. The experience of the reader of the paper as to the rarity of tabes among negroes had probably been duplicated by most of those present. At one time he had been a firm adherent to the view that syphilis was the chief cause of tabes, but now he was inclined to think that it was absurd to assume that spinal-cord diseases must necessarily be due to one cause. This had been impressed upon him by a study of multiple sclerosis. He had recently had a group of cases of traumatic tabes, without any evidence of syphilis. In his experience, the Argyll-Robertson pupil had been present first in by far the larger number of cases; next the pain; thirdly the loss of the deep reflexes. He had found hypotonus to be a remarkably early symptom. In at least three cases seen in the past month this symptom had been present when the other symptoms had been so indefinite that he had been in doubt as to the diagnosis of tabes. His later experience had led him to conclude that only a small percentage develop general paresis, but sufficiently often to lead to the suspicion that they are both due to the same factor. Whenever he found that several of the cardinal factors developed at one time he made an unfavorable prognosis, and he had learned that the cases in which the symptoms develop asymmetrically were those in which the disease progressed least rapidly. His experience confirmed the published statement of Dr. Collins, that treatment of syphilis does not seem to prevent tabes. On the other hand he believed that the progress of tabes could be stayed by vigorous antisyphilitic treatment.

Dr. Edward D. Fisher said that the statistics in the paper gave a fairly good picture of tabes. In his experience the earlier symptoms had been slight pains of a rheumatic character, associated with ataxia. About this time, or shortly afterward, the Argyll-Robertson pupil usually developed. Very few of his recent cases had shown optic paralysis, although it had been much more common a few years ago, thus showing the great variations in individual statistics.

Dr. Joseph Collins said that he had recently analyzed the symptoms of tabes occurring in 100 consecutive cases. Out of this num-

ber there had been four negroes. Two of his negro patients had been males, and two females. Out of the 100 cases, 7 were women, and five of these had been under thirty years of age. Three of these women were actresses, all of them apparently syphilitic. He had made his analyses without any preconceived notions regarding them. There was a definite history of syphilis in 63 cases, and an indefinite one in 17, bringing the possible relationship of sexual infectious disorder to tabes up to 80 per cent. In his experience the bladder symptoms and numbness of the lower extremities had been the more common initial symptoms. Next to these came pain, and next in order the giving way of the legs. This latter symptom had been very commonly met with. Of the 100 cases of tabes seen in the last three years, rectal crises had not been observed in a single one. He was becoming more and more convinced that orthodox and thorough anti-syphilitic treatment had not the slightest effect, either in delaying the appearance of tabes or mitigating its severity, and this seemed to him a most potent argument against tabes being a parasyphilitic disease.

Dr. Charles L. Dana said he had made an analysis of fifty private cases of tabes dorsalis that he had had under his care during the last two or three years. They represented, of course, a more intelligent class than those seen in the hospitals and clinics, and, consequently, the facts obtained from them were more trustworthy and valuable. Among those fifty there were forty-nine men and one woman. The ages at which the disease began were as follows: Under 20, none; between 21 and 30, inclusive, 15; between 31 to 40, inclusive, 15; between 41 to 50, inclusive, 20; between 51 to 60, inclusive, 3.

These figures showed that the critical period for the development of the disease was about the fortieth year of life, and, as a matter of fact, very few cases develop after that time, or, at least, after the forty-fifth year.

Twenty-eight, or over one-half of the patients were native born. He had notes of Cubans, Frenchmen, Italians and Mexicans, afflicted with the tabes, but none of negroes.

The number of cases in which there was an admitted venereal lesion of some kind, was 34, making a percentage of 68; and this was the highest percentage he could obtain, even with the most liberal interpretation. It is a common occurrence to hear patients state that they have had a venereal lesion many years before, but had absolutely no secondary symptoms, and among the 34 who admitted a lesion, 10, or one-third, denied positively ever having had any secondary symptom; while 10 others admitted that they did have such symptoms, and the other 14 were not very sure whether they had had them or not.

Of those who had specific lesions, 20, or about two-thirds were treated with more or less vigor for syphilis, while the remainder received little or no treatment.

Dr. Dana had investigated his statistics again, with reference to the influence of antisyphilitic treatment in preventing the development of tabes. This is a point, which, as many know, was studied with a great deal of thoroughness by Dr. Collins and himself several years ago, and so far as the figures were concerned, they were led to conclude that the treatment, even the most thorough, seemed to have very little effect upon the subsequent development of the tabetic process.

Studying these 50 cases independently, Dr. Dana found that among those who were carefully treated, after having received the initial lesion, the tabes developed twelve and a half years later. Among those who received no treatment at all, the disease developed, on an

average, seventeen years later. He would not draw any other inference from this than that, in all probability, the treatment of the primary disease does not seem to postpone the development of the tabes.

Among his 50 cases, there were 3 with Charcot's joints, or 6 per cent., and these all occurred in the early stages of the disease. In three cases paresis subsequently developed.

Of the 50 cases there were 7 who showed some evidence of a knee-jerk. In three the knee-jerk was present on one side only; in two it was slow or delayed, and in one it was present only on reinforcement. In two it was exaggerated. In one case the knee-jerk after having been abolished for four or five years, returned, remained present for two years, and again disappeared.

Gastric crises were present in four cases. Dr. Dana had seen the gastric crisis, as the initial symptom, last for several years before the other symptoms of the malady were recognized.

Tachycardia was, in his experience, a quite frequent phenomenon in tabes dorsalis, but actual heart lesions were more rare.

Hemiplegia occurred in two cases, early in the disease. Paraplegia occurred in one case, also early in the disease; and in these cases restoration of function was nearly complete. Facial paralysis, a rather unusual phenomenon, occurred in one case.

As to the initial symptoms, that most complained of, when the patients were first seen by Dr. Dana, was pain, in 15 cases; or both pain and ataxia in 10 cases. Among other early symptoms were sensations of great weariness (3 cases), rectal crisis (3 cases), persistent insomnia, attacks of vertigo, impotence, optic atrophy (2 cases). The Argyll-Robertson pupil should, perhaps, be classed among the most common of the early symptoms.

Dr. Dana found that many patients were either commercial travelers, or were men who had been upon their feet a great deal, or had indulged in excessive dancing. One of his patients had traveled twenty thousand miles in nineteen years. Two cases began after a severe fall or accident, and one after what was considered a sun-stroke.

The most important point in connection with the study of the symptoms of tabes, was that of the relation of the early symptoms to the prognosis. The outlook for patients suffering from this disease was extremely different in different individuals, as we know; some tolerated it marvelously well, while in others the nervous centers seemed to yield very rapidly. He did not venture to attempt any generalization regarding his own experience upon this point.

Dr. Thomas, in closing the discussion, said that he had only examined systematically for hypotonus in the last three years. He had been disappointed at not finding it more frequently. His statistics of the initial symptoms had been compiled from the histories rather than from the examinations. In most of his cases of optic atrophy the ataxia had been slight.

DEMONSTRATION OF MISS FLORENCE SABIN'S MODEL OF THE MEDULLA, PONS AND MID-BRAIN,

WITH REMARKS UPON THE VALUE OF WAX RECON-
STRUCTION AS A METHOD OF STUDYING THE
NERVOUS SYSTEM.

Dr. Lewellys F. Barker, of Baltimore, Associate Professor of Anatomy in the Johns Hopkins University, made the demonstration. The method of reconstruction, he said, origi-

nated with the embryologists in a desire to show in plastic form what they studied in sections. His, of Leipsic, many years ago, introduced his method of graphic reconstruction. The method of wax reconstruction directly from serial sections was introduced in Breslau in 1876. This method, with some modifications, had been employed by Miss Sabin, the constructor of the model to be demonstrated. Before beginning work one must have a faultless series of sections, an intelligent idea of the contents of those sections, a suitable mechanism for the projection of the sections magnified, some degree of spacial sense, and a certain amount of mechanical skill, together with a vast deal of patience. The model represented the medulla, pons and a portion of the mid-brain of a human fetus near full term. Miss Sabin's work was begun a year ago last March. The drawings were obtained by the projection method, and these were then transferred to wax plates, which were then superimposed.

Dr. Herter moved a vote of thanks to Dr. Barker, Dr. Thomas and Miss Sabin for their remarkably interesting and instructive contribution. This was seconded, and carried unanimously.

23. EIN BEITRAG ZU DEN PRIMÄREN COMBINIRTEN SYSTEMERKRANKUNGEN IM KINDESALTER (A Contribution to Primary Combined Systemic Diseases in Childhood). Hans Luce (*Deutsche Zeitschrift für Nervenheilkunde*, 12, 1897-1898, p. 68).

A child born of healthy parents first showed signs of disease when it was five years old. Weakness of the legs, disturbance of speech, spasticity of all the extremities, tonic contraction of the muscles after slight irritation, absence of muscular atrophy, were the most interesting features of the case. There had been neither infectious disease nor traumatism to explain the development of these symptoms. Death occurred nine months after the beginning of the disease.

The spinal cord presented a combination of the lesions of amyotrophic lateral sclerosis and tabes dorsalis. The crossed pyramidal tracts were partially and symmetrically degenerated from the proximal portion of the pons to the sacral region. In the right direct pyramidal tract the degeneration could only be traced into the upper thoracic region, and in the left it was still less intense. The cells of the anterior horns, especially those in the lower thoracic region, were much fewer than normal, and greatly diseased, and yet the anterior roots were only slightly degenerated. Usually in amyotrophic lateral sclerosis, the cells of the cervical region are most affected. The degeneration of the sensory portion of the cord was like that seen in tabes (degeneration of the posterior roots, of Lissauer's zones, of the postero-median root zones, of the posterior horns, of the fibers of Clarke's columns, and of the spinal roots of the ninth and fifth nerves). Important changes were found in the fibers of the projection and association areas of the cerebral cortex. Inflammatory foci were not observed anywhere, and the case was one of primary systemic disease.

The writer thinks he has demonstrated the possibility of the existence of tabes dorsalis, of amyotrophic lateral sclerosis, and of a combination of both diseases in childhood. The importance of the case therefore is apparent.

SPILLER.

Periscope.

CLINICAL NEUROLOGY.

24. THE OCULAR PHENOMENA IN GENERAL PARALYSIS OF THE INSANE. W. R. Dawson and D. F. Rambaut (British Medical Journal, 1898, ii., Sept. 10).

The authors record a series of observations made on this disease in the Richmond Asylum, Dublin, on some forty cases. In 92% of the cases the pupils were unequal, but the authors state that since this condition is by no means unknown in healthy people, too much stress should not be put upon it. In twenty-three cases the size of the pupil was recorded. Eight had mydriasis in both eyes and six myosis; three had myosis in one eye. In one case myosis and mydriasis were found, each in one eye. Reflex iridoplegia was noted in two mydriatic and three myotic cases. The Argyll-Robertson pupil was found five times in one eye and three times in both. Out of thirty cases examined for fundus changes, three showed advanced atrophy, five, optic neuritis. A certain amount of variation was to be expected in the ocular symptoms as a patient might show inequality, contraction, or dilatation or irregularity of outline at one time and not at another. Loss of light reflex or accommodation reflex might be present for weeks and then disappear and these reflexes be found normal.

25. ZUR PATHOGENESE DER HERDERSCHINUNGEN BEI DER ALLGEMEINEN PARALYSE DER IRREN (The Pathogenesis of Convulsive Seizures in General Paresis). W. Muratow (Monatschrift f. Psychiatrie u. Neurologie, 3, 1898, p. 40).

Muratow first calls attention to two classes into which the cerebral symptoms naturally fall. In the first of these there is the steadily progressing dementia with well localized brain symptoms, secondly those cases in which epileptoid and apoplectic attacks occur with circumscribed affection of sensibility and defects of movements. The pathogenesis of this second class of cases presents particular points of interest since the convulsive seizures introduce a new element in the disease and somewhat deflect the gradually developing clinical picture. It was with the idea of gaining some light on these questions that the material of the Preobrajensky Asylum was studied.

In the post-mortem examination of 123 patients with general paresis dying during the past four years, the authors shows that in every case there was a diffuse peri-encephalitis with atrophy of the cortex. In nearly all the cases the frequently described changes in the arachnoid and the ependyma were noted. In one case circumscribed softening was found, and in eight cases there was inflammation of the dura, but in neither of these conditions was there any appreciable relationship to apoplectic attacks. In no cases were there evidences of sudden bleeding, though many of the cases died in the convulsive seizures. In five cases with local convulsive seizures microscopical studies were

made. In all of these there were extensive inflammatory and degenerative changes in the cells of the motor area in addition to changes in the blood vessels and lymph spaces. Degeneration and disappearance of the tangential and radial fibers were also noted. On the evidence given by pathological anatomy there would seem to be no structural differences which give rise on one hand to localized convulsive seizures in cortical epilepsy, or on the other to those in general paresis. The difference must be in the cell activities which may be influenced by the changes in the encephalic inflammatory processes. The constant tremor is due, the author holds, to degeneration of the pyramidal tracts. The dysarthria is probably associated with a cortical ataxia of the frontal lobes. The changes in muscular sense are dependent upon degeneration of the tangential fibers. In common with the views of Nissl and of Weigert, the author states that the parenchymatous changes in the ganglion cells are of primary origin and are not dependent upon fiber degenerations, though their further degeneration is intimately connected with such other changes.

26. ANATOMIE PATHOLOGIQUE ET ÉTIOLOGIE DE LA PARALYSIE GÉNÉRALE (Pathological Anatomy and Etiology of General Paresis). Discussion. *Ann. méd.-Psychologique*, 7, 1898, pp. 440, 448, 464.

A lengthy discussion of the French medico-psychological society is here to be found on the etiology and pathology of general paresis. The main points of contention were regarding the occurrence of general paresis in the young, Régis presenting a paper on this subject, while Christian took a stand against its occurrence as also against any meta-syphilitic interpretations from the standpoint of the etiology. In the matter of the syphilitic etiological factor the prevalence of syphilis among the Arabs and their comparative immunity from general paresis was much in evidence. The discussion should be read to be thoroughly appreciated.

27. SUR DES LÉSIONS SPINALES DANS LA PARALYSIE GÉNÉRALE (On Spinal Lesions in General Paresis). G. Anglade (*Arch. de Neurologie* 6, 1898, p. 81).

The writer has described the pathological lesions in 20 cases which were clinically studied in Foncaill's Thèse de Paris, 1898. In the spinal cord there was found in all of the paralytic cases, degeneration of the exogenous fibers of the posterior and lateral columns; the direct cerebellar tract, the direct pyramidal and Gowers' tracts being involved less often. In the gray matter of the cord, there was fiber degeneration and chromatolysis of the cells in the anterior and posterior horns and in Clark's columns. Leptomeningitis, increase in the neuroglia, arterio-sclerosis and phlebitis were also found.

In the non-paralytic cases the writer finds much the same types of cell changes and he states that many of the lesions are similar to those found in syphilis, though most writers on the pathology of this disease state otherwise. He propounds a theory bearing on the pathogenesis of these changes.

28. CASUISTISCHE BEITRÄGE ZUR DIFFERENTIAL-DIAGNOSE ZWISCHEN LUES CEREBRI DIFFUSA UND DEMENTIA PARALYTICA NEBST EINEM ANATOMISCHEN BEFUNDE (Clinical Contribution to Differential Diagnosis of Diffuse Cerebral Syphilis and General Paresis, with an Autopsy). C. Winckel (*Arch. f. Psychiatrie*, 30, 1898).

Although this long report does not permit the drawing of definite conclusions, it is interesting on account of the wealth of detail of the

observations. The general conclusions from a clinical standpoint are as follows: In the six observed cases syphilis is certain. In three instances where the dates of infection were obtainable, mental disorders first appeared after a lapse of several months, 13, and 20 years respectively. In these cases specific treatment had been carried out beforehand, it was pursued in the institution with a more or less favorable result both as regards physical and mental symptoms in four cases, and with absolutely no result in the 6th case, which terminated in general paralysis. Heredity was to be noted in 2 cases.

The clinical picture of the psychical symptoms is most variable. After a premonitory period (5 cases) in which an alteration of character was remarked, maniacal excitement and anxiety and depression, euphoria and apathy succeeded each other or predominated. In two instances hypochondriasis existed, in another auditory hallucinations. Three cases manifested lessening of intelligence to a moderate degree, and without any progressive tendency, one patient was entirely cured, and two others in whom the picture resembled more nearly general paralysis went on to progressive dementia with delusions of grandeur.

Physical symptoms. Ocular manifestations; transitory paralysis of 3d and 6th pairs; symptom of Graefe unilateral, in two cases. In all the cases except one, absence of consecutive disappearance of the light reaction of one or both pupils; this symptom may be a relic of a former syphilis. Permanent or transitory aphasia, paresis of the limbs or face, 4 cases. Tendon reflexes were found normal once, different on the two sides, twice and exaggerated three times. Hypoalgeria in two cases. Ataxia was marked in one case, barely perceptible in another, disorders of articulation permanent or temporary, and more or less precocious in all.

Duration of the disease from $3\frac{1}{2}$ - $4\frac{1}{2}$ years, and even longer in one instance.

Several of the symptoms are also to be found in general paralysis, eye troubles, disorders of articulation, of sensibility and of the reflexes. As means of diagnostic importance are to be noticed, the variable character of the ocular affections, the aphasia, the temporary paresis, the non-progressive nature of the intellectual weakening, the co-existence of specific eruptions, and the favorable effect of specific treatment (even resulting in recovery) and the prolonged duration of the trouble.

The autopsy of the sixth case revealed a wide spread endarteritis of the arteries of the base; besides this lesion which is decidedly syphilitic, the other appearances approach those of general paralysis, cellular infiltration of meninges and vessels, variable atrophy of the fibers, adhesions of pia mater to the cortex, predominance of these lesions in the anterior lobes, absence of focal lesions.

It is the opinion of the author that it is secondary atrophy of the tangential fibers that produced the terminal symptoms resembling those of general paralysis, the circulatory changes due to the endarteritis and perivascular infiltration explaining the earlier mental and physical symptoms, such as changes in disposition, mental deterioration and apoplectiform attacks with paresis and aphasia.

29. LES PARALYSIES GÉNÉRALES PROGRESSIVES (General Paresis). Dr. Klippel (*Archives generales de Médecine*, 1898, p. 641).

The author speaks of the complications which have arisen in the study of general paresis due to the great variation in the clinical types of the disease. He proposes here a classification of the cases along three lines: (1) Primary inflammatory general paresis; (2) Secondary

or associated general paresis, and (3) Degenerative general paresis at times specific. These large groups include among others the cases of alcoholism presenting the paralytic syndrome; the cases of syphilis with sclero-gummatous lesions of encephalitis so often included under the general head pseudo-paresis.

I. In the first group the characteristic lesion is simple inflammatory exudation in the sense of Cohnheim. This produces a diffuse sclerosis which bears a close relationship to the primary parenchymatous inflammation.

The cells of the cortex are rapidly destroyed. Others soon become swollen and present very fine granulations. The nuclei are granular, and show the picture of degeneration rather than that of atrophy. By the Golgi method there is seen to be a marked diminution in the number of gemmules. The symptomatology of this group includes the gradual loss of the faculties, motor, sensory and physical in the inverse order of their evolutionary development.

II. In this group the same lesions and the same symptomatology are secondary to chronic alcoholic poisoning, to cerebral atheroma, to idiocy and to certain forms of the insanity of puberty.

III. In the third group, the character of the pathological lesion is different. Simple inflammation may be entirely absent, and in its place there may be found a number of tissue changes. Tuberculosis of the meninges in a chronic form, diffuse sclerotic gummata, atheroma of the base of the brain are the common lesions.

As a further complication the author holds that a certain amount of overlapping may occur, and that in this group more particularly, as a result of various toxic agents, the inflammatory type of cell changes may supervene.

The author further speaks of the spinal forms which according to their method of onset take on the lateral sclerotic, amyotrophic, bulbar or neuritic types. These indicate according to the author only that the general paretic process is susceptible of certain predominant localizations in some one part of the nerve axis, and these localizations give rise to the clinical pictures of the myelopathic diseases, but considered from the standpoint of their evolution, their relationship to the general paretic process is evident. In a further subdivision of his subject he points out the relationship of the symptomatology to disease of the visceral organs. These visceral lesions include, 1, pre-existing lesions of tuberculosis, alcoholism and arteriosclerosis; 2, those secondary to degenerations, from nephritis, broncho-pneumonia or from arterial diseases. He believes that there is some relationship between such visceral lesions and changes in the lymphatic centers of the cerebrospinal axis. In conclusion the author insists upon the need of a closer analysis of the pathological pictures; there has heretofore been too much combining of disease processes which are evidently separated by many years of evolution, it may be, at times; thus a diffuse inflammatory process may succeed an arterio-sclerotic lesion which in its turn was induced by the alcoholic poison. The relationship of bacterial poisons should also be taken into account.

Book Reviews.

JOURNAL OF MENTAL SCIENCE.

With the first number of 1899 this well established journal presents itself in a new cover, and with a larger size page and a new style of typography. We believe it to be a marked improvement on the old time form and style and welcome it in its new dress.

ARCHIVES GÉNÉRALES DE MEDECINE.

Improvement in form and typography would seem to be the fashion, and this journal falls into line in a new style which is also a marked improvement on its former make-up. This excellent journal is to be congratulated upon its departure, and its subscribers cannot but enjoy the change.

ZEITSCHRIFT FÜR AUGENHEILKUNDE. S. Karger, Berlin, 1899.

No apology is needed in this place for noticing this new journal to be issued from the press of S. Karger of Berlin, since the relationships of neurology and ophthalmology are so close. This journal promises to be a particularly desirable addition to the list of journals devoted to this specialty. It is to be edited by Professor Kuhnt of Königsberg and Professor V. Michel of Würzburg, and has a number of collaborators whose reputations are certainly continental if not international. It hopes to pay especial attention to the neurological symptomatology in eye diseases.

ARCHIVES OF NEUROLOGY AND PSYCHOPATHOLOGY. States Hospital Press. Utica. 1898.

With the year 1898 the second volume of the States Hospital Bulletin is brought to a close, and the publishers announce a new title for this well-known bulletin with an increase in its scope. The present number contains two articles; one, to us, a fanciful and utterly impossible sort of a theory of neuron energy, designated new, and a reprint of Dr. Van Gieson's lengthy and interesting report to the State Commissioners in Lunacy. We hope for a regular issue of this new quarterly and trust to review more at length the contents of the first and second numbers of the year. JELLIFFE.

ARBEITEN AUS DEM GESAMMTGEBIET DER PSYCHIATRIE UND NEUROPATHOLOGIE, von R. v. Krafft-Ebing. III. Heft. Johann Ambrosius Barth, Leipzig, 1898. Mk. 4.50.

Several miscellaneous essays of this well known author are here brought together in convenient form. They include: 1, The Etiology of Paralysis Agitans; 2, Waking and Dream States; including such states in epilepsy, in neurasthenia, hysteria and alcoholism; 3, Typical Delirium in Epilepsy; 4, An Idiopathic Periodic Recurring Insanity in the form of a Delirium; 5, Epileptic Psychoses; 6, Surgical Treatment of Epilepsy; 7, Ecmmesie; 8, On Retrograde General Amnesia; 9 and 10, Some Hysterical Conditions.

Some of these studies are new and some are reprinted from prominent neurological journals. The collection is one of interest.

JELLIFFE.

UEBER DIE SEXUELLEN URSACHEN DER NEURASTHENIE UND ANGSTNEUROSE, von Dr. F. Gattel, Nervenarzt in Berlin. August Hirschwald, Unter den Linden 68. 1898.

The treatment of sexual matters is becoming more prominent of recent years and much good will be derived from a careful study of this most important life function. There is still room for accurate determination of normal standards in this regard and the present brochure presents its conclusions in a decidedly one sided manner. From a study of one hundred cases at the Vienna clinic the author would lead us to believe that masturbation or other sexual irregularity was the main cause of all neurasthenic woes. That such *are* powerful factors needs no corroborative evidence, but as the *causae verae* we are not yet in a position to positively affirm. The character of a patient's sexual habit is just as important as that of his gastro-intestinal canal or urinary habit, and is worthy of a more careful clinical study, and the author's small pamphlet is full of interesting details which merit reading.

JELLIFFE.

LEHRBUCH DER PSYCHOPATHOLOGISCHEN UNTERSUCHUNGSMETHODEN von Pr. D. R. Sommer in Giessen. Mit 86 Abbildungen. Urban und Schwarzenberg. Berlin and Vienna, 1899. 10 marks.

"That a workman may be known by his tools" is an adage applicable in clinical medicine as well as in the carpenter shop. Instruments of precision have been the guides to most of our refined methods of analysis and were it not for such helps the interpretation of many clinical studies would be impossible. The present volume is to be thoroughly recommended. It offers much new material in the way of the more exact measurements of nervous processes both peripheral and central, and is an index of the newer and more thorough paths that the alienist and psychiatrist must tread to be *en rapport* with the times.

In four sections the author discusses the general methods of the more exact clinical investigations of nervous and mental diseases. Part I deals with optical methods; Part II., Motor graphic methods for determining changes in movements. Investigations of the reflexes, direct motor expression; Part III., Registering by acoustic methods. Use of the phonograph in the analysis of speech irregularities, motor graphic methods of studying speech defects, etc., and Part IV., Investigation of the psychical functions, including time reactions, quantitative determinations of inner processes, perception and conception, orientation, memory, association, ability to count and compute, etc., etc.

The work is thorough and well printed and will undoubtedly be a valuable guide for those interested in the careful study of nervous and especially mental cases.

JELLIFFE.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

SOME POINTS OF SPECIAL INTEREST IN THE STUDY OF
THE DEEP REFLEXES OF THE LOWER EXTREMITIES.¹

I. ANKLE-CLONUS WITH ABSENCE OF KNEE-JERK; 2. THE SIGNIFICANCE OF ANKLE-CLONUS IN THE DIAGNOSIS OF HYSTERIA FROM ORGANIC DISEASE; 3. PATELLAR CLONUS; 4. TENDO-ACHILLIS JERK IN TABETICS.

BY CHARLES K. MILLS, M.D.,

PROFESSOR OF MENTAL DISEASES AND OF MEDICAL JURISPRUDENCE IN THE
UNIVERSITY OF PENNSYLVANIA; NEUROLOGIST TO THE PHILADELPHIA HOSPITAL, ETC.

ANKLE-CLONUS WITH ABSENCE OF KNEE-JERK.

The patient, H. J., white, laborer, was admitted to the men's wards of the Philadelphia Hospital, September 12, 1898. On admission he was found to be paralyzed in both legs. He had great difficulty in breathing, complained of pain in the precordial region and of a band of pain encircling the body between the ensiform cartilage and the umbilicus. His family history showed nothing of importance. When fifteen he had had rheumatism, but otherwise up to the time of his present illness he had been healthy. He admitted the moderate use of both alcohol and tobacco.

About five weeks before his admission to the hospital he began to be troubled with constipation and difficulty in expelling his urine. The bladder would become distended and painful, and it was only with great difficulty that he could urinate. One week previous to his admission he began to have a feeling of numbness in both feet, this gradually extend-

¹ Read before the New York Neurological Society, Jan. 3, 1899. For discussion on this paper see p. 172.

ing upwards. Five days before admission he went to bed with no evidence of paralysis, but on attempting to rise the following morning he found that his legs were paralyzed, the precordial and girdling pain developing about the same time, while breathing became so difficult that he had to assume a sitting position.

Examination on admission showed him to be a man of good muscular development; but with an appearance of being poorly nourished. His face had a waxy look, lips and finger tips were bluish and cold, and dyspnea was marked. His tongue protruded straight without tremor; it was pale and anemic looking, tooth-marked at the edges and coated with a dirty white fur. His pulse was rapid, small and water-hammer in character.

The normal heart sound was nowhere heard. At the mitral area two loudly blowing murmurs were heard, the louder being systolic and transmitted toward the axilla. At the aortic cartilage two more murmurs were heard; the one, diastolic, was soft and blowing; the other, systolic, was harsh and rasping. The latter was plainly transmitted into the vessels of the neck. Pressure over the upper part of the abdomen caused pain.

When admitted the foot of the right leg could be lifted about six inches from the bed when the leg was extended, but in the left this movement could not be carried out, and when it was attempted the leg flexed at the knee and the heel dragged. The toes of both feet moved freely. When catheterized immediately after admission the urethra was found to be hyperesthetic, and while passing the catheter both legs and thighs were quite forcibly flexed, whether voluntarily or because of reflex action was not definitely determined. It was learned that the patient had had no headache, that his appetite had been very poor, and for a few months he had had some cough. The pupils were equal and reacted both to light and in accommodation, and no paralysis of any of the extraocular muscles was present at the time of admission or later. At no time had he any paralysis or anesthesia of the upper extremities.

He was examined by me three hours after admission. He did not, and apparently could not, make any movements with the right leg, which seemed to be completely paralyzed, and was semi-flexed at the hip and knee. On the anterior, inner and outer aspect of both knees and on the dorsum of the foot were dusky reddish areas.

Examination for sensation showed that the patient was analgesic over the right thigh and front of the leg below the knee, and on the dorsum of the foot to its inner edge. Tactile sensation was preserved, as was also the thermal sense. In

general terms the sensory phenomena in the left lower extremity were the same in distribution as in the right, but a little less extended over the dorsum of the foot and great toe. A glance at the diagram (Fig. I) gives a correct idea of the extent of the analgesia, and a comparison of this with one of Heiberg's plates (Fig. II) shows that the areas chiefly affected were those supplied by the anterior crural, the communicans peronei, and the musculocutaneous or superficial peroneal nerve.



FIG. I.

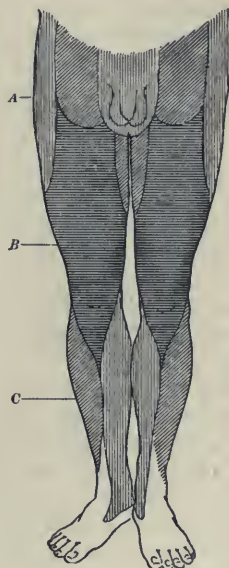


FIG. II.

FIGURE I. Area of analgesia in the patient with lost knee-jerk on both sides and ankle-clonus on one side.

FIGURE II. Sensory nerve areas of the anterior surface of the lower extremity: A, external cutaneous (cutaneus femoris lateralis); B, crural; C, external saphenous (communicans peronei); musculocutaneous (unshaded area on dorsum of foot).

The most interesting clinical point in connection with the examination of this man was that brought out by a study of the muscle and tendon phenomena in the lower extremities. Quadriceps-jerk on each side was prompt and marked; a striking fact in connection with the intense degeneration of the muscle subsequently found, and additional evidence that the muscle-jerk is essentially different from the knee-jerk. Both knee-jerks, for which he was frequently tested, were absent. Ankle-clonus was undoubtedly present on the left side; some-

times it was quickly exhausted, but at other times it persisted; it was never absent at any of the examinations which were repeated several times during three days. Ankle-clonus and toe-jerk were both absent on the right side, and toe-jerk was not present on the left. Both elbow-jerks were absent.

Most of the clinical phenomena above noticed were determined on September 12, the day of the patient's admission to the hospital. At 7 P. M. of this day he seemed to be worse and complained of severe pain in the small of the back and over the precordium. He passed a comparatively comfortable night, but did not sleep any. On the morning of September 13, he said that he felt much better and would like to sit up in his chair. The action of his heart was slower and steadier, and the bluish color of the lips and finger tips had disappeared. The color of his face was also better. He had one stool containing mucus. He slept quite well during the night of September 13th, but early on the morning of the 14th he had a slight attack of dyspnea. He passed a very uncomfortable night on the 14th; dyspnea was extreme. The action of the heart was forcible and rapid, notwithstanding the continuous use of digitalis. The dyspnea continued all day. He complained greatly of precordial pain. He died at 3.40 A. M. on the 15th.

The autopsy showed the body of a well developed man. Post-mortem rigidity and lividity were marked, and the feet were slightly edematous. A considerable quantity of clear, light yellow fluid was present in the peritoneal cavity. The surface of the liver was smooth, and its edges were well marked. On section the knife met with little resistance, and the cut section was mottled and fatty. On section the pulp of the spleen was quite friable. The suprarenal capsules were normal. The right and left kidneys showed similar conditions. The fatty capsule was thick; the fibrous capsule stripped with difficulty, leaving a rough surface in areas. On section the cortex was found to be narrowed. The substance of the kidneys was quite firm. The right and left pleural cavities contained about 500 c. c. of fluid.

The left lung on section showed marked edema and at its apex a slight consolidation. It also showed a slight anthracosis. Similar conditions existed in the right lung. Much subpericardial fat was present. The heart muscle, particularly around the left ventricle, and more especially the papillary muscles, showed marked fatty change. Both the aortic and mitral valves were sclerotic, the aortic admitting two fingers and the mitral four.

Careful examination of every part of the brain showed no gross lesion. Considerable fluid blood was found in the

vertebral canal external to the dura, but not more than is often seen in spinal autopsies. No organized clots were found at the level of any of the lumbar segments or elsewhere in the canal. The spinal membranes were not thickened and showed no signs of either diffuse or focal meningitis. No gross lesion of any of the spinal nerves in their intraspinal course could be detected.

The entire spinal cord was put in formalin for hardening. Portions of one of the posterior spinal roots from the first lumbar segment, and also parts of the anterior crural nerve on each side were removed, and placed in a one per cent. solution of osmic acid. Other portions of the anterior crural nerves were placed in Müller's fluid. The pieces of nerve were taken from just below Poupart's ligament. A fragment of muscle was taken from the same region and was put in Müller's fluid.

Segments from the first lumbar region and lower levels of the spinal cord were embedded in celloidin, cut and stained by the methods of v. Lenhossék and Weigert, by acid fuchsin, and by Delafield's hematoxylin. Sections were also stained by Marchi's method.

The microscopical examination of the specimens was made in the William Pepper Laboratory of Clinical Medicine, of the University of Pennsylvania, by Dr. William G. Spiller, who has furnished the following report:

"The sections from the spinal cord stained by the above methods appear to be perfectly normal. The cells of the anterior horns are as numerous as in normal cords, and stained by the method of v. Lenhossék show the usual arrangement of the chromophilic elements and the central position of the nucleus. No traces of hemorrhage can be found in the lumbar region, and the vessels are not distended to an unusual degree. The anterior and posterior spinal roots stained by acid fuchsin and the hematoxylin of Weigert seem to be normal—that the posterior, however, are not *absolutely* normal is shown by the osmic acid. No degeneration of the lateral columns can be detected by the method of Weigert or of Marchi.

"A portion of one posterior root taken from the first lumbar segment and stained by osmic acid shows many fibers in which the myelin has assumed a beaded form, and probably more such fibers than a normal posterior root contains; this root, therefore, cannot be said to be *perfectly* normal, and yet the changes are perhaps too unimportant to explain any of the symptoms.

"Portions of one of the anterior crural nerves stained with osmic acid and "teased" present many fibers in which the

myelin has become irregularly beaded and others in which the myelin stains faintly with osmic acid.

"Sections from the anterior crural nerves stained with acid fuchsin and Delafield's hematoxylin appear to be nearly normal, each nerve fiber containing an axis cylinder, but when the hematoxylin of Weigert is employed certain bundles seem to contain fewer nerve fibers than is normal, and also an unusual number of small nerve fibers. These sections were made at a considerable distance from the peripheral terminations of the nerves.

"Section from the piece of muscle removed present a high degree of alteration of muscular tissue. When stained by Marchi's method, which is most valuable for recent degeneration of muscle, numerous fibers are found both in longitudinal and transverse sections filled with an excessive number of small black dots representing a fatty degeneration (Fig. III). In transverse sections the numerous rounded, greatly swollen muscular fibers, that have undergone hyaline degeneration and vacuolation, are most striking. Some of these fibers are very large, perfectly round, stain a deep red with acid fuchsin, and present a glassy appearance (Fig. IV). When stained by Van Gieson's method these swollen fibers are of a dark wine color. The sarcolemma nuclei have in general disappeared from those muscular fibers that are entirely degenerated. The interstitial connective tissue nuclei are not very excessive. The blood vessels of the muscles are normal. Intramuscular nerve bundles contain fibers each with an axis cylinder when the acid fuchsin is used, and by the hematoxylin of Weigert the medullary sheaths are stained black, although possibly less intensely so than in normal muscles.

"In longitudinal section the blocks of hyaline degenerated muscles are exceedingly numerous, and quite frequently one is able to trace one of these swollen blocks without a trace of transverse striation, into a normal fiber transversely striated containing sarcolemma nuclei, and of much smaller caliber than the hyaline block in which it originates (Fig. V). In some of these swollen hyaline muscles the interior is more faintly stained than the exterior. Numerous spaces in certain of these hyaline fibers resemble vacuoles. The absence of proliferation of the connective tissue and of atrophied muscular fibers is very striking, and the process was evidently a recent one. Many of these hyaline blocks in longitudinal sections present longitudinal striation, and in some the peculiar arrangement of the hyaline tissue in irregular bands is seen, as described by Marinesco."

The findings in this case of lost knee-jerks with presence of ankle-clonus may be briefly stated. The spinal cord appears

to be perfectly normal; a posterior root from the first lumbar segment stained with osmic acid exhibits a somewhat unusual number of nerve fibers with beaded myelin; the anterior crural nerves seem to be distinctly degenerated when the osmic acid is employed, but the changes are slight as shown by Weigert's hematoxylin and the acid fuchsin; the muscular tissue is greatly altered and many of the muscular fibers are tumefied and present a high degree of hyaline or fatty degeneration.

In the light of the clinical history and pathological findings, what is the most probable explanation of the unusual syndrome—absent knee-jerk with ankle-clonus on one side—presented by this case? During the patient's life the most probable diagnosis seemed to be a focal lesion involving the lumbar cord in that region through which the reflex arc for the patella is completed, namely, the lumbar segments from the second to the fourth inclusive. One objection to this diagnosis was, however, to be found in the distribution of the analgesia, which was not confined to the area supplied by the crural nerve, derived from the lumbar cord and plexus, but also included the areas for the communicans peronei and musculocutaneous (superficial peroneal of Heiberg), which are branches of the external popliteal, and, therefore, derived from the sacral cord and plexus. The existence of heart disease with both mitral and aortic murmurs lent color to the view that the lesion might be one or more foci of softening in the cord. Another theory that seemed tenable was that a lesion like a hemorrhage involved some of the extramedullary or intramedullary lumbar roots, or the cells of the anterior horns, and compressed the lateral columns of the cord in the upper or midlumbar region, but against this view was the improbability of a localized hemorrhage causing the irregular distribution of the analgesia, although, of course, an irregular clot, an exudate, or multiple clots might produce almost any unusual grouping of symptoms. As has been stated in the history of the autopsy and microscopical examination, the most critical investigation revealed no lesion of the substance of the cord or of the intramedullary nerve roots in the lumbar region, though one of the first lumbar posterior roots examined was not entirely normal. The blood found in the

vertebral canal was fluid and only such as is frequently seen in spinal autopsies, the leakage from the tissues wounded in removing the posterior portions of the vertebral column. Fluid blood could not have produced the symptoms, for gravity would have caused the blood to descend to the lower part of the spinal column, and ankle-clonus would then hardly have been obtained.

The findings in this case give us an explanation for absent knee-jerk and presence of foot-clonus not hitherto afforded, in fact I know of no similar case with necropsy. The muscular tissue examined was greatly altered, presenting marked hyaline and fatty degeneration, while the anterior crural nerves were partially degenerated, as was also the posterior root examined. I attribute the lost knee-jerks conjointly to disease of the muscles and to the degeneration of the crural nerves, alterations in muscle and nerve tissue affording a comparatively easy explanation of the loss of the patellar reflexes.

It is not improbable that the difficulty in voiding the urine and feces, of which the patient complained, was due to a degeneration of the muscles of the abdominal walls, similar to that in the piece of sartorius examined, as the contraction of the abdominal muscles plays an important part in the evacuation of bladder and rectum. I cannot regret sufficiently that muscle and nerve tissue were not taken from widely separated portions of the body and submitted to careful microscopical examination. The symptoms presented by the patient were those which most neurologists would attribute to disease of the spinal cord.

We must seek to find the cause of this muscular degeneration, which was of an intense degree in the piece of sartorius examined. It seems not improbable that the cardiac trouble may have been the cause. The absence of atrophied fibers and of proliferation of the connective tissue, and the presence of recent degeneration, as shown by the method of Marchi, are in conformity with the short duration of the paralysis in the lower limbs. We know that muscles undergo hyaline degeneration as a result of acute infectious disease, but I have been unable to obtain the history of any such affection in my patient. Muscular degeneration may be more common

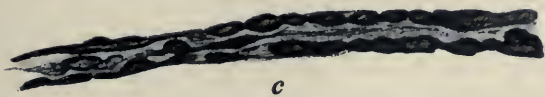
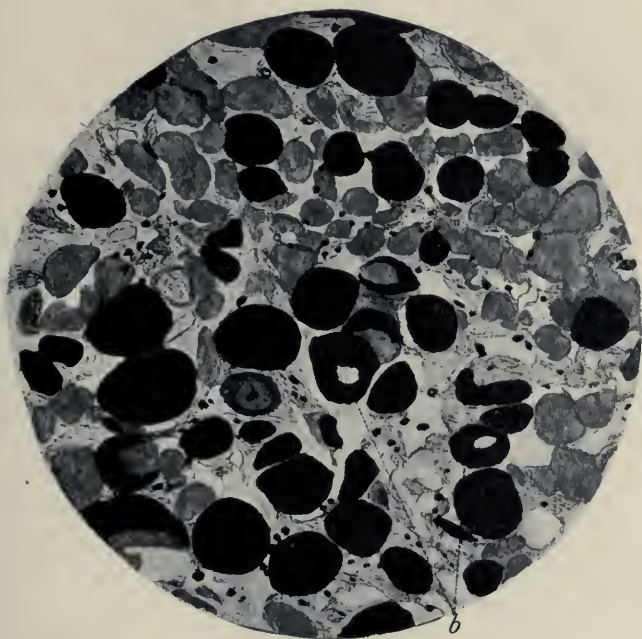


FIG. IV. Transverse section of the sartorius muscle stained with acid fuchsin. The small unaltered fibers measure about 30 microns, while the larger ones that have undergone hyaline degeneration (represented in black in the drawing) measure 70 to 100 microns. Some of the fibers show vacuoles (*b*). (*c*) Degenerated fibers from one of the anterior crural nerves. The myelin sheaths are irregularly beaded.

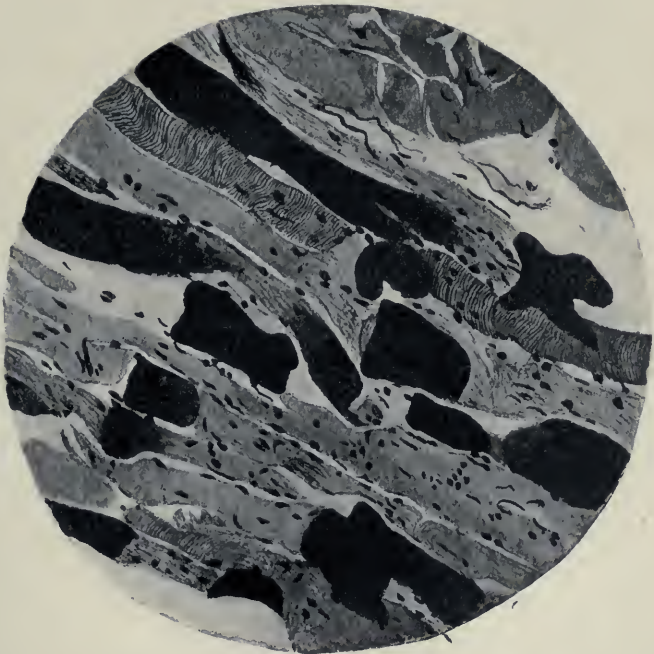


FIG. V. Longitudinal section of the sartorius muscle. The hyaline masses which were round when viewed in transverse section (Fig. IV.) are long and irregular in Fig. V. In many muscular fibers the transverse striation is lost, though in some it is preserved. Some muscular fibers terminate in hyaline masses.

than we suppose when the heart has been acting feebly for a long time.

This is the day for the theory of autointoxication. It may be that we have to seek the cause of the muscular degeneration of this case in the imperfect elimination of toxic substances from the system, brought about by an impaired circulation. Whatever theory is preferred, the fact must be accepted that the sartorius muscle of my patient presented signs of intense degeneration, probably of recent origin, and that the degeneration of the muscles was apparently much greater than that of the nerve fibers. We should be cautious, however, in accepting a degeneration of nerve fibers which appears slight under the microscope, as of little importance. Nerve cells and nerve fibers probably suffer great impairment of function before they undergo structural change, and apparently slight degeneration of nerve tissue may cause severe functional disturbance.

The explanation of the distinct and persistent ankle-clonus on one side is not easy, indeed remains somewhat in doubt, and is worthy of discussion.

While it is well known that in multiple neuritis the tendon reflexes are often absent at some stage, and especially early in this disease, exaggerated knee-jerks have been observed. Usually as the disease advances the reflexes diminish, and in the majority of cases disappear. Even ankle-clonus has been observed in rare instances in multiple neuritis.

In one case of multiple neuritis, with the symptoms of a comparatively mild form of erythromelalgia, I have even observed slight patellar clonus on one side at one of the examinations, but this could not be elicited subsequently. This was a man twenty-five years old, studied in my wards at the Philadelphia Hospital, three weeks after the onset of the disease.

What is most significant in its bearings on the case under discussion is that v. Bechterew³ in one case of multiple neuritis observed ankle-clonus after the patellar reflex had disappeared.

With these facts before us it is possible that the unilateral

³ v. Bechterew, W., *Neurologisches Centralblatt*, 1895, p. 1157.

ankle-clonus present in my patient was due to a neuritis attacking the nerve branches to the tendo Achillis. In multiple neuritis the degree of inflammation and degeneration may vary at different times in different nerve trunks and branches, and it is probable that if inflammation was present in this case in the nerves to the tendo Achillis the disease was here in an early stage.

It may be that the muscles and nerves of the calf were not involved in the degenerative process, and the absence of analgesia over the posterior and inner aspects of the leg makes this view not improbable.

Some cases of lost knee-jerk with presence of ankle-clonus in which inflammation of peripheral nerves is seen may be explained on the theory that the cells of the sacral region controlling the ankle phenomenon are thrown into a state of hyperexcitability by the inflammation of nerves having their reflex arc in the lumbar segments concerned with the patellar reflex.

Another explanation is to be found in the fact that while it is uncommon to find exaggerated reflexes in neuritis, it has been shown by Weir Mitchell⁴ that certain forms of irritation of a nerve trunk may cause excessive irritability in the muscles supplied by the affected nerve, as was seen when a frozen nerve was thawed.

It is scarcely necessary to an audience of neurologists to direct attention to the anatomy and physiology of the knee-jerk and of ankle-clonus, and yet it may be of some little service to do this as a part of the discussion of this unusual pathological grouping of the two most important of the deep reflexes of the lower extremities, especially as it is not yet firmly established through which segments of the lumbar and sacral cord these reflexes are consummated. In the knee phenomenon the tendon of the patella having been struck, the excitation is carried to the spinal cord by crural sensory fibers, and enters by the dorsal roots into a certain portion of the dorsal column, and thence passes to the dorsal horn; next it takes its course through the intermediate gray substance until it reaches the cells of the ventral horn; and thence

⁴ Mitchell, S. W., *Injuries of Nerves and Their Consequences*, p. 60.

the motor excitation goes through the motor roots and crural nerve to the anterior muscles of the thigh. It does not matter for the purposes of our discussion whether knee-jerk is or is not a pure reflex, or whether, as is most probable, it is due first to direct stimulation of the muscle, and secondly to reflex influence; in any case absence of this phenomenon follows a complete break in any portion of this path. In considering the loss of knee-jerk in this as in any other case, therefore, it should be remembered that it may be abolished from disease of any of the following parts: the peripheral sensory nerve fibers, the posterior roots, either in their extramedullary or intramedullary portion, the gray matter through which the reflex collaterals of the posterior roots pass, the cells of the ventral horns, the motor roots, the motor nerve fibers, or the extensor muscles of the leg.

According to Westphal's investigations, when the patellar reflex was abolished the part of the spinal cord affected was in the transitional region from the thoracic to the lumbar cord. Many later researches have placed the spinal segments for this reflex at a lower position of the cord, and according to different observers it would seem that the reflex arc might be situated anywhere from the second to the fifth lumbar segment.

Pineles⁵, in describing a case which he designates as one of sacral tabes with lost knee-jerks, says the first normal fibers of the posterior roots, in examining the cord from below upward, were observed in the midlumbar region, and from here upward the root entrance zones were gradually filled with normal fibers. Redlich⁶, says that the patellar reflex arc is now believed to be a little lower than Westphal placed it, and that usually it is located about the second lumbar segment. Another case of sacral tabes has recently been reported by Auerbach⁷. The knee-jerk was absent on each side. The degeneration by microscopical examination seemed to be in the first and second sacral and the lower lumbar posterior roots.

⁵ Pineles, Arbeiten aus dem Institut für Anatomie und Physiologie des Centralnervensystems an der Wiener Universität, Heft IV., p. 341.

⁶ Redlich, E., Die Pathologie der tabischen Hinterstrangserkrankung, p. 100.

⁷ Auerbach, Deutsche Zeitschrift für Nervenheilkunde, V. XI., p. 143.

Higher up the posterior root fibers were normal. In the middle portion of the lumbar region the posterior roots were practically intact. This case is therefore similar to the one reported by Pineles.

In a case of tumor of the cauda equina, reported by Dejerine and Spiller⁸, knee-jerks were lost, and all the posterior roots were degenerated as high as, but not including, the first lumbar roots. The knee reflex must be lower than the first lumbar, as indicated by this case, for the examination was made by the method of Marchi, which would have revealed any recent degeneration in the first lumbar roots if it had been present, and did reveal intense degeneration of the roots below the first lumbar.

From these and other investigations it is at least clear that the segments of the spinal cord included in the patellar reflex arc are situated somewhere between the second and fifth lumbar segments inclusive. The facts before us would seem to indicate that it is probably located where it has been placed by Edinger⁹, Starr¹⁰ and others, namely, in the second or third lumbar segments, or both.

The anatomy of the Achilles reflex arc has not been so thoroughly studied as that of the patellar reflex arc; in fact I have not at command any investigations definitely locating the segments of the sacral cord concerned in this reflex. Some clinical and anatomical considerations would, however, indicate that the segment of the cord concerned in this reflex is probably situated where it has been placed by Starr and others, namely, somewhere from the first to the third sacral segments inclusive. Paralleling the description above given of the patellar reflex arc and its lesions, it would therefore follow that loss of the Achilles reflex would occur from a destructive lesion affecting the peripheral sensory nerve fibers from the tendo Achillis, the dorsal roots of these fibers, the gray matter

⁸ Dejerine and Spiller, *Comptes rendus de la Soc. de Biologie*, 1895, and personal communication by Dr. Spiller.

⁹ Edinger, L., *Vorlesungen über den Bau der nervösen Centralorgane des Menschen und der Thiere*, fünfte Auflage, Leipzig, 1896, p. 313-314.

¹⁰ Starr, M. A., *The Diagnosis and Localization of Spinal Cord Diseases*, in *A System of Practical Medicine by American Authors*, ed. by Loomis and Thompson, V. 4, 1898, p. 71.

through which the reflex collaterals of these roots pass, the cells of the ventral horns, the motor roots, the motor nerve fibers to the gastrocnemius and soleus muscles, or these muscles themselves.

It is not enough to know the particular segment or segments of the cord concerned in the patellar reflex. Only certain portions of these segments are involved in the reflex arc, and it therefore follows that lesions affecting the spinal cord at the proper *level* to include the reflex arc might, if limited to certain transverse regions of the cord, permit the reflex act to be consummated. Pineles¹¹ says that Westphal's thorough investigations have clearly shown that when the patellar reflex is absent a certain zone in the spinal cord must be diseased. This zone is limited by an imaginary line parallel to the posterior septum, drawn through the point where the posterior horn makes a bend; by the inner side of the posterior horn and by the periphery of the cord. This zone was called by Westphal the root-entrance zone (*Wurzeleintrittszone*). Westphal's investigations referred only to the transitional region from the thoracic to the lumbar cord, but it is probable that an area in one or more of the lumbar segments somewhere from the second to the fourth inclusive, corresponding to Westphal's zone, contains the patellar reflex arc. A lesion outside of this area, and outside of the other portions of the spinal cord included in the reflex arc, might be present without any disturbance of the patellar reflex. A focal lesion like a hemorrhage or small focus of softening, or a disease like syringomyelia, might involve the segments of the cord at the level which corresponds to the patellar reflex arc without abrogating the patellar phenomenon, a fact which must be borne in mind in the explanation of some exceptional cases of lumbar focal disease. Such a case of retained patellar reflex in a case of tabes, for example, has recently been reported by Achard and Lévi.¹² The root-entrance zones were comparatively intact, although the posterior columns were much degenerated.

¹¹ Pineles, *Arbeiten aus dem Institut für Anatomie und Physiologie des Centralnervensystems an der Wiener Universität*, Heft IV, p. 341.

¹² Achard, C., and Lévi, L., *Nouvelle iconographie de la Salpêtrière*, 2, 1898, p. 83.

In order to show at a glance the mechanism of the patellar and the ankle phenomena I have had made a diagram which shows both the patellar reflex arc and the Achilles reflex arc,

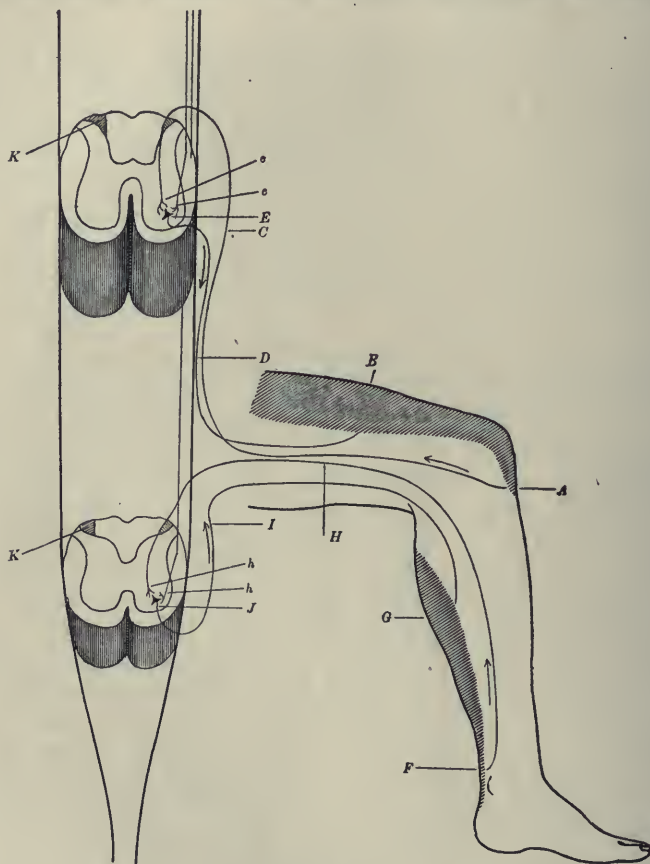


FIG. VI.

FIGURE VI. Scheme of the patellar and the tendo-Achillis reflex arc: A, patellar tendon; B, quadriceps muscle; C, sensory (afferent) fiber from the patellar tendon; D, motor fiber from cell in the ventral horn to quadriceps muscle; E, cell-body in ventral horn; F, tendo Achillis; G, gastrocnemius muscle; H, sensory fiber from the tendo Achillis; I, motor fiber passing from cell in the ventral horn to gastrocnemius muscle; J, cell-body in ventral horn; K, area corresponding to Westphal's zone; the direction of the arrows shows the direction of the sensory and motor impulses.

and the controlling tracts related to these arcs. A brief study of this diagram (Fig. VI) will show that a destructive lesion in-

volving the lumbar segment or segments in which the patellar reflex arc is included might abrogate the patellar reflex on both sides, and at the same time by involving the corticospinal tract to the sacral cord remove the cerebral inhibition from the Achilles reflex and thus allow the foot phenomenon to coexist with lost knee-jerk. A glance at this diagram will also show how advanced disease involving the nerve and the muscle forming portions of the patellar reflex arc would destroy the patellar reflex, the Achilles reflex arc remaining, and this perhaps by less advanced and irritative nerve disease being stimulated until foot-clonus is manifested. It will also be seen from a study of this diagram that limited focal disease of the cerebral cortex, or of the cerebrospinal tract, might cause increased clonus on one side, while peripheral disease of crural nerves or quadriceps muscle might destroy the patellar reflex. Other possible explanations which will be given of lost knee-jerk with ankle-clonus can also be better understood by a study of the diagram.

Let me next glance at the literature of the subject of lost knee-jerk with presence of ankle-clonus. This is limited. I have been able to find the record of only ten cases, although, of course, it is not improbable that some have been overlooked. In Sternberg's¹³ monograph reference is made to seven cases. I have notes of three other cases, which, including my own, make eleven in all. The cases being so few, a summary of the most important of them will, I am sure, not be considered out of place. Bechterew (l. c.), as already indicated, in one case of multiple neuritis has observed ankle-clonus after the patellar reflex had disappeared.

Dr. F. X. Dercum, in a personal communication to the writer, has given the details of the case of a patient who, after falling from a height, striking on his buttocks, became paraplegic. At the time of examination this patient could flex his thighs slightly on his abdomen, but had no control over his feet. Marked wasting of some of the muscles of the lower extremities was present, and the man had blebs or blisters over his toes, ankles and feet. He made considerable improvement

¹³ Sternberg, M., *Die Sehnenreflexe und ihre Bedeutung für die Pathologie des Nervensystems*, Leipzig und Wien, 1893.

while under Dr. Dercum's care, but remained partially paralytic. He presented no evidence of local injury to the spinal column. The conditions as to sensation were not recalled by Dr. Dercum. In this case knee-jerk was lost on both sides and ankle-clonus was present on both sides.

Erb¹⁴ has recorded a case in which the reflex clonus and the biceps tendon reflex were observed, but the patellar reflex and the reflex of the adductors was in no way to be obtained. This was one of three cases of compression of the spinal cord with kyphosis. The kyphosis was in the lumbar region, the first and second lumbar vertebræ forming the point of the kyphosis.

It will be recalled by members of the New York Neurological Society that Dr. Joseph Fraenkel¹⁵, at the March meeting of this year, presented a child two and a half years old, in whom the knee-jerks were absent and ankle-clonus was present on both sides. The child, born by the breech after a difficult labor, had abnormal lower extremities from birth, and was not, at the time of presentation to the society, able to sit, walk, or stand. Electrical examinations showed extensive degeneration of all the muscles except those of the calves. Dr. Fraenkel seemed, from his remarks, to incline to the view that the case was one of poliomyelitis, or that the syndrome was the result of a dropsy, a focal hemorrhage, or a localized cavity formation. He explained the existence of the ankle-clonus by the theory that the calf-muscles were in a state of increased tonus, due to the fact that the antagonists were gone. Dr. Joseph Collins accepted the pathological explanation given by Dr. Fraenkel, but Dr. B. Sachs believed it better to assume that in the case there was some developmental defect.

At the meeting of the Berlin Society for Psychiatry and Nervous Diseases, held November 8, 1886, Mendel¹⁶ presented a man of forty with (1) loss of patellar reflex; (2) bilateral foot-clonus; and (3) paradoxical contraction in the left foot. This patient, who was not syphilitic, began in 1877 to have pains in the left hip, which varied in intensity and later were felt in the

¹⁴ Erb, W. *Archiv. f. Psychiatrie*, 5, p. 802.

¹⁵ Fraenkel, J., *Proceedings of the New York Neurological Society, Jour. Nerv. and Ment. Dis.*, V. 25, April, 1898.

¹⁶ Mendel, E., *Archiv. für Psychiatrie*, V. 19, pp. 524-525.

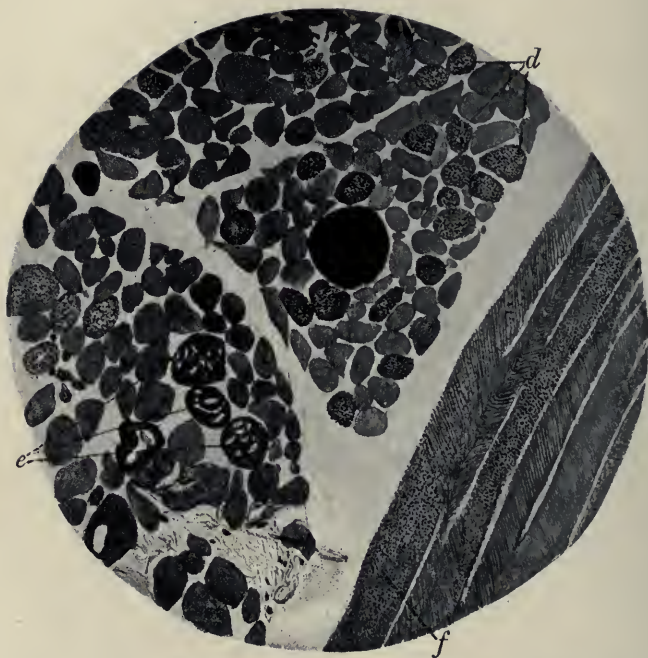


FIG. III. On the left of the drawing is a transverse section of muscular fibers stained by Marchi's method; on the right a longitudinal section stained by the same method. Many fibers are filled with numerous black dots (*df*) representing fatty degeneration of the muscular fibers. Some of the fibers show vacuoles (*e*). In the center of the picture is a large fiber that has undergone hyaline degeneration.

right leg and shoulder. The legs became weaker while the arms remained intact. In the lower limbs considerable disturbance of cutaneous sensation was found and the muscle sense was impaired. The muscles of the thigh and calf were atrophic, especially on the left side. The electrical reactions could not be taken. The left pupil was larger than the right. The left half of the face was not as well innervated as the right. Mendel believed the case was one of multiple sclerosis, and that a focus was located in the reflex arc of the quadriceps muscle; that another was more deeply seated, and in the lateral column, involving the sciatic (control) tract. One of these foci caused loss of the knee-jerk, the other caused foot-clonus. Remak had examined the electrical conditions of this patient previously, and found a great diminution in the response of the left crural nerve, indications of reaction of degeneration in the quadriceps extensor and no contraction of the anterior tibial muscle from irritation of the left peroneal nerve. He believed that a circumscribed transverse myelitis was present in the upper part of the lumbar swelling, and that this on the left side had involved the cells and caused paralysis in the crural territory, with implication of the anterior tibial. Oppenheim said he had often seen cases with dorsal clonus of the foot with Westphal's sign (loss of knee-jerk), for example, in a case of vertebral injury. It seems to me probable that Oppenheim may not have fully considered his words when he made the sweeping assertion that he had *often* seen foot-clonus with lost knee-jerk.

In connection with Mendel's explanation of the syndrome in this case it may be remarked that Buzzard¹⁷, in a discussion on the clinical significance of the deep reflexes before the Medical Society of London, stated that the occurrence of ankle-clonus in a leg which presented at the same time a total absence of knee-reflex, not infrequently pointed to a disseminated sclerosis.

Fleury¹⁸, in a case of typhoid fever examined on the twelfth day of the disease, found the tendon reflexes (presum-

¹⁷ Buzzard, Brit. Med. Jour., Nov. 7, 1885, V. 2, p. 868.

¹⁸ Fleury, Note sur les rapports de la trépidation épileptoïde du pied avec l'exagération des réflexes rotuliens, in Revue de Médecine, quatrième année, 1884, V. 4, Paris, p. 656.

ably from the context the patellar reflexes) absolutely abolished. While this was the case, he obtained on the contrary at the first attempt a very marked trepidation in the right as well as in the left foot. The tendon reflexes remained abolished during the entire time that the patient was in the hospital. The trepidation also continued, but presented notable variations. Fleury concluded that epileptoid trepidation does not always coexist with exaggeration of the patellar reflexes, and that it can exist in subjects in whom these reflexes are normal, impaired or abolished.

Regarding the probable lesions in a case which presents the unusual syndrome of lost knee-jerk with presence of ankle clonus, several conclusions may be drawn from a study of the case here presented and an examination of the meager literature of the subject:

(1) The syndrome may be due to a compression or destroying lesion, such as caries with pachymeningitis, or transverse myelitis, involving the spinal cord in the region of the patellar reflex arc, namely, somewhere between the second and fifth lumbar segments, and most probably about the second or third lumbar segments.

(2) The syndrome may be due to disseminated sclerosis, foci of sclerosis being present both in the reflex arc for the patella and in the lateral column.

(3) The syndrome may be due to focal lesions like hemorrhage, softening, or cavity formation, attacking points in the reflex arc and also the lateral columns.

(4) The syndrome may be due to peculiar forms of developmental arrest of the spinal cord, as for instance, to defect in the gray matter of the lumbar segments and in the lateral columns.

(5) The syndrome may be due to a combination of muscular and neural disease, as in my case, and as was probably also the case in the man suffering from typhoid fever as recorded by Fleury. It is known that hyaline degeneration of muscular fibers occurs especially in typhoid fever. Fleury's case was probably in its pathology not unlike my own.

(6) On theoretical grounds it seems probable that the syndrome might be due to a focal lesion in the cerebral cortex, or in the cortical spinal (pyramidal) tract, or to arrested de-

velopment of the tract, associated with disease (inflammation or degeneration) limited to the crural nerves and their muscles.

THE SIGNIFICANCE OF ANKLE-CLONUS IN THE DIAGNOSIS OF ORGANIC FROM FUNCTIONAL DISEASES, ESPECIALLY HYSTERIA.

The second subject connected with the study of the deep reflexes of the lower extremities to which, in accordance with the announcement of this meeting, it is my purpose to direct attention is one which some of those present may at first sight regard as so far settled as not to be worthy of special discussion; that this, however, is not the case is forced upon me by several considerations. The discussion of this subject at the Philadelphia Neurological Society has brought out a striking difference of opinion among neurologists of large experience, a fact familiar to those who have read the proceedings in the *JOURNAL OF NERVOUS AND MENTAL DISEASE*.¹⁹ I have not much to present in addition to what has already been published in these proceedings, but believing the subject to be one still worthy of close discussion I have concluded to include its presentation in this paper, hoping to get decided expressions of opinion from the members of the New York Neurological Society.

The majority of neurologists, both in this country and abroad, so far as I have been able to note their opinions, seem to disagree, in part at least, with Gowers and the writer regarding ankle-clonus in hysteria and other functional affections. v. Bechterew²⁰ says that many neurologists still share the opinion of Gowers, although cases of pure hysteria with ankle-clonus have been observed. Bechterew says also that he has seen ankle-clonus, exactly like that occurring in organic disease, in cases of myoclonus multiplex, and also he has seen it in cases of akinesia algera. Oppenheim²¹ believes that both foot-clonus and paradoxical contraction occur in hysteria.

¹⁹ Proceedings of the Phila. Neurological Society, *Jour. Nerv. and Ment. Dis.*, v. 24, Nov., 1897.

²⁰ Bechterew, W., *Neurologisches Centralblatt*, 1895, p. 1157.

²¹ Oppenheim, H., *Archiv. für Psychiatrie*, v. 19, pp. 524-525. (In the discussion of a paper presented by Mendel to the Berlin Society for Psychiatry and Nervous Diseases, Nov. 8, 1886.)

Sternberg,²² in his monograph on the tendon reflexes, says that in about twenty per cent. of hysterical cases foot-clonus is present.

As my own views are practically in accord with those of Gowers,²³ let me recall these. While not denying the possibility of a true uniform clonus in hysterical paraplegia, Gowers holds that it is so rare that it does not materially lessen the value of this sign of organic disease. "Certain facts," he says, "might be stated with confidence. 1. In many cases the myotatic irritability was perfectly normal. 2. In others there was distinct, though slight excess, insufficient to give a true clonus. 3. In hysterical contracture, and depending on it, there might be a clonus like that which occurred in health in standing on 'tip-toe.' 4. A 'spurious foot clonus' was common, depending on a voluntary contraction in the calf muscles, pressing down the foot, and varying in degree from time to time, the clonus varying with it. This was very characteristic and was a most important diagnostic sign of hysterical paraplegia.

"Apart from the slight clonus produced through a voluntary depression of the foot of the patient in response to the passive flexion of the ankle, and readily recognized, and the true clonus which may be obtainable during hysterical contracture, a true foot clonus or a rectus clonus deserves the greatest weight, as all but conclusive evidence of organic disease. I have known many mistakes in diagnosis," he says, "in which lateral sclerosis was mistaken for hysterical paraplegia owing to disregard of the evidence afforded by this symptom, but I have never known the opposite error from undue regard to this symptom. Moreover, an excess of myotatic irritability in so-called hysterical paralysis must depend on more than functional disease. There must be changes in nutrition, and consequent persistent defective control of the muscle reflex centers."

What I believe may be justly designated a large clinical experience, extending over many years, leads me to subscribe fully to the views thus expressed.

²² Sternberg, M., *Die Sehnenreflexe und ihre Bedeutung für die Pathologie des Nervensystems*, p. 254.

²³ Gowers, Sir W. R., *Brit. Med. Jour.*, Nov. 7, 1885, v. 2; and *A Manual of Diseases of the Nervous System*, v. 1, 2d ed., Phila., 1892, p. 451.

If I am right in following Gowers in these expressions of opinion, the most important sources of error in those who maintain the contrary are (1) the nonrecognition of the coexistence of organic lesions, and especially focal lesions and hysteria in the same case; (2) the misinterpretation of cases in which toxemia or malnutrition are more important factors than hysteria; and (3) absolute errors of diagnosis.

The fact that organic disease, and especially a focal lesion, either cerebral or spinal, is often associated with hysteria, and sometimes even induces grave hysterical manifestations, may mislead even the skilful diagnostician with regard to ankle-clonus. In the cases reported to the Philadelphia Neurological Society by Spiller²⁴ and by Burr, this combination of organic disease and hysteria seemed to me a source of error. In Spiller's case the patient was paralyzed in his right arm and to a less degree in the right leg, had intention tremor in the right limbs, sensation on the right side had been partially lost but had been regained, knee-jerk and quadriceps-jerk were exaggerated in the right lower extremity, positive and persistent ankle-clonus was present on the right side only, other tendon and muscle phenomena were also exaggerated on the right side, chin-jerk was present, and some amnesia for language was an interesting feature. Most of those who took part in the discussion of this case leaned to the view that it was one of hysteria, although to my mind it was undoubtedly one of organic hemiplegia. The disappearance of the anesthesia was not inconsistent with the view that it may have been a pressure phenomenon due to a lesion of the posterior limb of the left internal capsule. The double hemiplegia may have been apparent rather than real, and even the history so carefully given is susceptible of other explanation than that of hysteria. At the most, I believe that a critical study of the entire case shows it to be one of grave organic disease probably associated with grave hysteria. In the case recorded by Burr,²⁵ the patient was a hemiplegic of a somewhat common motor type, with great

²⁴ Spiller, W. G., An Unusual Case of Hemiplegia, *Jour. Nerv. and Ment. Dis.*, v. 24, No. 7, July, 1897.

²⁵ Burr, C. W., A Case of Hemiplegia (Possibly Hysterical) with Ankle-Clonus, in the Proceedings of the Philadelphia Neurological Society, *Jour. of Nerv. and Ment. Dis.*, v. 24, No. 11, Nov., 1897, p. 707.

exaggeration of all the reflexes, including persistent ankle-clonus; but in addition, this man had a number of attacks which seemed to be clearly hysterical with motor, emotional and sensory manifestations of a marked character. At times he had complete left hemianesthesia, at other times anesthesia of the segmental type. The case was probably organic, with hysterical epiphenomena. Seguin²⁶ believed that hysterical phenomena were much more frequent in left than in right hemiplegics.

With regard to the misinterpretation of cases in which toxemia or pernicious malnutrition plays a part, I would only say that the diagnosis of pure hysteria is the matter of vital importance. Hysteria of pure and typical form is a disease already so large in its manifestations as not to require us to attribute to it symptoms due to other causes. As held by Gowers, profound nutritional disorders may be present in hysteria as in organic disease, but the symptoms due to these nutritional changes are not in a strict sense a part of the hysteria. A patient suffering from syphilis or from alcoholism may have either hysteria or neurasthenia, but it is syphilis with neurasthenia, syphilis with hysteria, or perhaps hysteria or neurasthenia the direct resultant of the poisonous influence of syphilis or of alcohol; and it is necessary that the clinician should separate phenomena due to the toxemia and those due to hysteria, which is fundamentally a cerebral (psychical) affection.

Among the forms of organic disease with ankle-clonus in which I have seen absolute errors of diagnosis made are brain tumor, hemorrhage or softening of the brain, disseminated sclerosis, so-called lateral sclerosis, ataxic paraplegia, syphilitic spastic paraplegia, transverse myelitis, and caries with pachymeningitis. In some of these cases, when the incorrect diagnosis was made, sufficient time had not elapsed for a clear decision.

It must perhaps be admitted that in one class of grave hysterical cases, marked and persistent ankle-clonus is sometimes present, although even in these cases, as suggested by Gowers, the clonus may be secondary to the condition of contracture, or the myotatic irritability may be due to nutritional changes.

Rhein, at the meeting of the Philadelphia Neurological So-

²⁶ Seguin, E. C., *Opera Minora*.

ciety at which the report of Dr. Burr's patient was presented, spoke of a case which seemed from the history to be clearly hysterical, and in which the phenomena, ankle-clonus being among them, disappeared under hypnotic suggestion. The chief symptoms were spasticity in the legs, general tremor when walking, spasmodic strabismus, segmental anesthesia, and a true ankle-clonus, which persisted on one side but was easily exhausted on the other. This case upholds the view that true ankle-clonus may be present in cases with hypertonicity and the diathesis of contracture. An interesting medico-legal case, recently observed, lends confirmation to this view. Traumatic hysteria was certainly, in part at least, the true explanation, and in this case ankle-clonus of the most marked and persistent type was present. This patient had met with an injury in attempting to board a car. Her chief subjective symptoms were great pain in the head and left side, insomnia and general nervousness. Examination showed marked paralysis of the right upper extremity with partial loss of power in the lower, analgesia and thermal anesthesia over the right half of the body and limbs. She was unable to walk, stand erect or (apparently at least) to hold up her head. Both lower extremities showed a frequent tendency to spasticity, and at times she had attacks of severe spasm in them. The knee-jerks were exaggerated, and ankle-clonus was present on both sides, but most marked and persistent on the right, which was the side of the anesthesia and paralysis. This, in very general terms, was the condition of the patient at the time of my first examination in October, 1897. She was last examined by me November 11, 1898, when I found present most of the conditions just stated. She was still partially paralyzed in both the upper and lower extremities on the right side, and sensation was still partially lost over a large portion of the same side of the body. The right leg tended to rotate outward at the hip, and she had some contracture both in the upper and lower extremities. Knee-jerks were still exaggerated, but ankle-clonus was absent, although the left leg became spastic when handled. In addition to the hysteria, a hemorrhagic or other lesion affecting the motor tracts may have been present, although the case was undoubtedly in large part one of hysteria.

In hospital and private practice and in medico-legal work, I have examined a very large number of cases of the kind usually classed under the head of hysteria, hystero-neurasthenia, or neurasthenia. Where the diagnosis has been clear, that is, when the existence of an organic lesion could with confidence be excluded, ankle-clonus of any type has been very rarely present, and the persistent form of ankle-clonus has always been absent—excluding from this class only a very few cases in which hypertonicity or the diathesis of contracture was present. I have examined for this point thirty medico-legal cases. In seventeen of these cases the diagnosis of traumatic hysteria or traumatic hystero-neurasthenia, and in thirteen that of traumatic neurasthenia was made. These cases were all examined for muscle-jerks, knee-jerks and ankle-clonus. In only three of the thirty was ankle-clonus of any type present, and in these it was of the abortive or short lived form; in one of the three it was of the spurious type.

In every record of a case presumably hysterical, in which it is stated that foot-clonus is present, a statement should be appended describing the type of clonus. The cases will be few, and the clonus will be of the abortive or spurious form, excepting from this statement a few cases with spasticity and contracture.

PATELLAR CLONUS.

In examining for patellar clonus one of the best methods of grasping and pushing down the patella is by means of the separated thumb and index finger, either using steady pressure or supplementing this with occasional strong pushing movements.

As I sometimes found it difficult to elicit patellar clonus, even when it was present and persistent, owing to the difficulty in keeping up a steady traction and at the same time supplementing this with light percussions, I have had made a simple instrument which might be called the *patella-tractor*, to assist in studying this clinical phenomenon. It consists of an oval-shaped, closed metallic ring with a shank, the whole being attached to a handle which affords an easy grasp. The upper segment of the metallic ellipse is covered with rubber slightly roughened on its inner edge so as not to so readily slip. In taking patellar clonus this ring of metal is slipped over the

patella, the handle of the instrument being grasped below the knee in such a manner that both downward traction and some pressure can be exerted. By pulling steadily downward on the patella tension can without difficulty be kept up indefinitely. (Fig. VII.)

For patellar clonus I examined in all one hundred cases. These cases were chiefly patients in the wards for nervous diseases of the Philadelphia Hospital and included the following diseases:

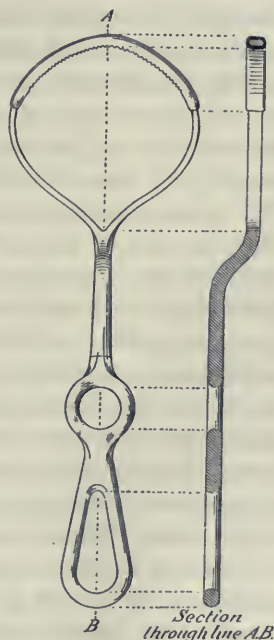


FIGURE VII. Patella-tractor.

Epilepsy, 10 cases; hemiplegia and monoplegia (from hemorrhage, thrombosis or embolism), 19 cases; cerebral syphilis, 4 cases; cerebrospinal syphilis, 3 cases; multiple cerebrospinal sclerosis, 5 cases; paralysis agitans, 4 cases; uremia, 1 case; cerebellar tumor, 1 case; chorea, 1 case; Ménière's disease, 1 case; chronic myelitis, 3 cases; syringomyelia, 1 case; progressive muscular atrophy, 3 cases; senility, 2 cases; functional tremor, 1 case; amyotrophic lateral sclerosis, 2 cases; ataxic paraplegia, 1 case; Friedreich's ataxia, 1 case; spastic paraplegia (probably syphilitic), 2 cases; multiple neuritis, 5 cases; tabes, 28 cases; general paralysis of the insane, 1 case; lead poisoning, 1 case; total, 100 cases.

Out of one hundred cases examined patellar clonus could be elicited in only seven cases. In two of these cases it was present, but only slightly marked; in one case, an old hemiplegic, it was slightly present on the paralyzed side; in the other, a case of senility probably with lacunæ of softening both in the brain and cord, it was slightly present on both sides. In a case of syringomyelia it was well marked on both sides. It was also well marked in two cases of cerebro-spinal syphilis. In one of these cases some paralysis was present in all four extremities, but most decided in the right arm. The patient had had no less than five paretic or paralytic attacks, and a history of syphilis six years before admission to the hospital. The lesions were probably of the nature of diffuse gummatous meningitis or meningomyelitis. Quadriceps-jerk, knee-jerk, gastrocnemius-jerk, ankle-jerk and the front-tap phenomenon were all plus. Patellar clonus was present on both sides, and the case was interesting because of the fact that while patellar clonus was present ankle-clonus was absent. In another case of cerebrospinal syphilis with well marked cerebral, cranial-nerve and spinal symptoms patellar clonus was present on the right side, but on the left was unobtainable, apparently because of strong contractures in the flexor muscles of the thigh. In this case also the quadriceps-jerk, knee-jerk, gastrocnemius-jerk and ankle-jerk and front-tap phenomenon were all present, but ankle-clonus was absent, although patellar clonus could be elicited on the right side. These two cases show that patellar clonus may be present in the absence of ankle-clonus, a clinical fact which would seem to call in question the statement that has been made that patellar clonus represents the highest grade of reflex excitability of muscle or tendon in the lower extremities. Ankle-clonus may be present and patellar clonus absent, and this is probably the most frequent clinical arrangement of these two phenomena; but patellar clonus may be present, as just shown, when ankle-clonus is absent. Reference has already been made to one case of multiple neuritis with symptoms of erythromelalgia, in which slight patellar clonus on one side was elicited at one of the examinations.

A study of the deep reflexes of the lower extremities, including patellar clonus, proved to be interesting in a case of

acute uremia admitted to the nervous wards of the Philadelphia Hospital during the time that I was engaged in examining the patients for most of the facts contained in this paper. This patient, J. S., 54 years old, was admitted to the hospital December 18, 1898. When admitted he was in a semi-conscious state; his pupils were contracted and did not react to light. His pulse was 50, fairly full and slightly irregular; temperature 99.04 F.; respiration 28; his right arm was paretic; both legs were spastic. His urine was one-sixth albumin and contained granular casts. A study of the deep reflexes of the lower extremities shortly after admission showed quadriceps-jerk on both sides marked, knee-jerk much exaggerated, especially on the right; gastrocnemius-jerk present; ankle-jerk present on both sides, but more marked on the right than on the left, a tap on the right tendo Achillis causing slight clonus; on the right ankle-clonus was present but not very persistent, on the left it was absent; patellar clonus was marked on both sides. Examinations on admission were made by my interne, Dr. Merritt. Twenty-four hours later, after the patient had been on active treatment for uremia, which consisted chiefly of hot packs, elixir of glonoïn and Bascham's mixture, all the tendon and muscle phenomena, including patellar clonus, were present, but were less marked than on admission. It is of passing interest to note in this case as in the last two mentioned that while patellar clonus was present on both sides, ankle-clonus could be elicited only on one.

Since taking up this subject anew I have not had the opportunity of examining any considerable number of functional affections for patellar clonus, but on analogy and on theoretical grounds I would be inclined not to expect patellar clonus in hysteria, neurasthenia and other functional nerve disorders, for the same reasons that I have given in stating my views with regard to the significance of ankle-clonus in the differentiation of organic from functional disease. It is not improbable that patellar clonus will be found in those cases of hysteria in which hypertonicity and the diathesis of contracture are present—cases of spastic and convulsive hysteria; cases of tetany, and some cases with choreic and athetoid phenomena.

v. Bechterew²⁷ says that patellar clonus is very common

²⁷ Bechterew, W., *Neurologisches Centralblatt*, 1895, p. 1157.

in cases of myoclonus multiplex, not infrequently the standing position alone being sufficient to throw the muscles of the anterior part of the thigh into clonic contraction.

TENDO-ACHILLIS JERK IN TABETICS.

Babinski,²⁸ before the Société Médical des Hospitaux de Paris, at the meeting held October 21, 1898, stated that in tabes most frequently the knee-jerk and the tendo-Achillis jerk are both abolished. Sometimes the alterations or disorders in the reflexes are on one side only, but the phenomena may be crossed. In rare cases the knee-jerk is abolished, and that of the tendo Achillis is normal. Finally in some cases the knee-jerk is normal, and that of the tendo Achillis is lost or impaired. Babinski thinks that the involvement or abolition of the reflex of the tendo Achillis is a sign as important in tabes as the sign of Westphal. He believes also that this reflex should be systematically examined, and that it is probable that in any of the cases of tabes where the patellar reflex is preserved, one can establish alteration of the reflex of the tendo Achillis.

Stimulated by this brief communication of Babinski, I have recently examined twenty-eight cases of tabes and tabulated the results. These results were, however, so uniform in character that they can be expressed in a few words. The ankle-jerk (tendo-Achillis jerk) was present in only three cases out of the twenty-eight. In one case the Achillis-jerk was quite marked on both sides. In this patient ataxia was present in both the lower and upper extremities. In a second case the Achillis-jerk was present but slight on both sides. In a third case the Achillis-jerk was not present except by reinforcement, when it could be brought out on the right side only. These three cases were all typical illustrations of tabes from three to nine years' standing, with sensory, cranial-nerve, and other well known phenomena. The quadriceps-jerk, knee-jerk, and gastrocnemius-jerk were present in all, and in one of the cases in which the Achillis-jerk was present the gastrocnemius-jerk was distinctly plus. Besides the twenty-eight cases of tabes, the other seventy-two cases in which patellar clonus was studied

²⁸ Babinski, *Le Progrès Médical*, 3e serie, T. 8. No. 44, Oct. 29, 1898, p. 301.

were also examined for tendo-Achillis jerk, and it was present on both sides in all cases.

In one case of tabes the study of the tendo-Achillis jerk was of decided value in reaching a correct diagnosis. This was a case sent to me for opinion by two physicians. The diagnosis of neurasthenia had been given, and the existence of tabes was regarded as in doubt, chiefly for the reason that both knee-jerks were present, although the patient had a recent history of what appeared to be shooting pains and some evidences of probable slight involvement of the bulbar nuclei. Ataxia was not present, but the tendo-Achillis jerk was lost on both sides—quadriceps-jerk, knee-jerk, and gastrocnemius-jerk being present. I concluded that the case was organic, probably a somewhat irregular form of tabes.

A study of the tendo-Achillis jerk may, therefore, be of considerable value in clinching the diagnosis in a doubtful case, and especially in a case in which the region of the patellar reflex arc in the cord has, up to the time of the examination, escaped in whole or in part. My study of the subject shows that the sacral cord is usually involved in cases of well marked tabes; but that the degeneration may in rare instances be confined for a considerable time to the sacral nerves and sacral portion of the cord.

Recently in a case of tumor of the cerebellum—a case presented to the Philadelphia Neurological Society, December 19, 1898, by Dr. William G. Spiller, examined in consultation by the writer, and operated upon later by Dr. John B. Roberts, a careful study was made of all the tendon and muscle phenomena. All the phenomena with which we are concerned in the present communication, quadriceps-jerk, knee-jerk, gastrocnemius-jerk and ankle-jerk were absent, as were also front-tap, ankle-clonus, and patellar clonus. A superficial examination of this case might have led to the diagnosis of tabes, and the case is of interest as pointing to one use to which the study of the muscle and tendon phenomena of the lower extremities in the differential diagnosis of encephalic tumor from tabes may be turned. When the *muscle* as well as the tendon phenomena are absent the diagnosis of tumor is more probable. What is the cause of the absence of these phenom-

ena in tumor cases? While their absence may be explicable on the theory of cerebellar influence, they are in some instances better explained on the theory of a more or less general toxemia excited by the growth. This toxic influence is, perhaps, in the first place exerted on the posterior roots of the spinal cord. It is well known that degeneration of the posterior spinal roots has been found in a certain percentage of cases of brain tumor. In all the twenty-eight cases of tabes studied during the preparation of this paper, both the quadriceps and gastrocnemius (muscle) phenomena were present, although in some of these it was apparently below the normal. Why should the muscle as well as the tendon phenomena be absent in these cases of brain tumor without direct involvement of the spinal cord and posterior nerve roots, while in the vast majority of cases of tabes the muscle response is preserved? It has occurred to me that the microscopical findings in the first case recorded in this paper may have some bearings on the explanation of this fact. It will be recalled that both muscle and nerve disease were distinctly present, and in fact the evidence of muscle degeneration predominated. The toxic influence exerted directly on the muscle may be the proper solution of the problem.

30. UEBER DIE INFANTIL-JUVENILE (FRÜH) FORM DER DEMENTIA PARALYTICA (On the Infantile Juvenile (Early) Form of General Paresis). G. Mengazzini (Monatschrift f. Psychiatrie u. Neurologie, 3, 1898, p. 53).

The author here describes one of the first if not the first case reported from Italy of general paresis of the young. The patient was twenty-one years of age. Her father had syphilis. The symptoms began at the age of sixteen with a gradual dulling of the mental faculties. She could no longer read, and then there began a progressive loss of power in her muscles with increasing dementia. Auditory and optical hallucinations were common. The deep reflexes were increased, pupils unequal, the right apparently immobile. There were no epileptic nor apoplectic attacks. She died of general exhaustion in profound dementia. The pathological findings confirmed the diagnosis. There was chronic meningo-encephalitis, atrophy of the frontal lobes and hemorrhagic internal pachymeningitis. Some interesting cord changes are also noted.

A CONTRIBUTION TO THE KNOWLEDGE OF THE STEREOGNOSTIC SENSE.

BY JOSEPH SAILER, M.D.,¹

ASSOCIATE IN THE WILLIAM PEPPER LABORATORY OF CLINICAL MEDICINE.

Excepting Hoffmann, almost no medical writer has seriously studied the stereognostic sense. It is not mentioned by Mills, Gowers, Brissaud, Oppenheim or Strümpell. Even in articles devoted to sensory phenomena, as those by Laehr, it has been wholly neglected, and the word does not occur in the index of the first thirty volumes of the *Archiv für Psychiatrie*. The following cases, therefore, are recorded, because in some respects they appear to throw light upon the sensory and psychic elements upon which this form of perception depends.

Case 1. B. A., male, aged 53 years, was admitted to the Presbyterian Hospital, February 24, 1897, suffering from left hemiplegia. He had a history of alcoholism. Although considerable improvement subsequently occurred, the patient is still a cripple, and requires constant attention. His present condition, December 7, 1898, is as follows: no facial paralysis; the left arm is weak, but capable of being moved in all directions; there is slight contracture of the muscles of the forearm, more pronounced in the flexors, so that the hand is habitually held half closed, there is almost no control over the movements of the fingers, but the patient can flex them until they almost touch the palms, and extend them again to their habitual position; the tendon reflexes and the muscle-jerks are all exaggerated. There is a moderate amount of resistance to passive extension of the hand.

Sensation shows the following peculiarities, tactile sense is perfectly normal, the cotton point being felt as readily and identified as promptly on the left side as on the right; localization of contact is exceedingly inaccurate, thus—if the hand is touched, the patient will indicate a point perhaps two inches away from the actual point of contact; if one of the fingers is touched, he is unable to recognize which one. Upon the forearm, he usually indicates a point about an inch above the actual spot. Above the elbow, however, his sense of localization is very good, and elsewhere the sense is well preserved, no essential difference being distinguished between the left and right sides. Recognition of the pain-sense is not, however, as prompt on

¹ Read before the Philadelphia Neurological Society, Dec. 19, 1898.

the left side. Temperature-sense is completely lost in the hand, neither hot nor cold is recognized, or, if either is guessed, the number of errors is greater than the number of correct results. Loss of temperature-sense in the forearm is also pronounced, but above the elbow the number of correct replies is in excess of the mistakes. The muscle-sense is lost in the hands. Trichosthesia, if this be a separate sense, is everywhere preserved.

Neither slight nor considerable alteration in position of the fingers appears to be perceived by the patient, ordinarily, he answers "up" and "down" alternately without any reference to the nature of the movements made. The same is true of motion at the wrist-joint. At the elbow-joint the answers are usually correct, although occasionally a mistake is made, even after a movement of considerable amplitude. If the patient is told to close his eyes, and the right arm is moved passively into some position, a not altogether unsuccessful attempt is made to reproduce that position with the left arm, and the patient is even more successful when told to imitate the position of the left arm with the right. A more careful investigation of tactile sensibility shows that the patient is unable to distinguish the number of fingers that touch the left hand. Change in the pressure is only noticed when very marked. The patient is unable in a majority of instances to distinguish between hard and soft substances. He has no perception of the shape of objects, nor can he guess their probable nature. The same objects placed in the right hand are recognized at once. A similar loss of tactile perception or *astereognosis*, is observed upon the forearm, that is to say, the patient is unable to distinguish accurately between hard and soft, or between rough and smooth surfaces. This perception is accurate on the right forearm. Of the sensory changes in the other parts of the body, the fine tactile sense is preserved on the face, pain-sense is preserved, but the recognition is somewhat delayed. Temperature-sense is lost to a point within an inch of the median line. Motion on the left side of the face is normal. The sensory phenomena in the left lower extremity are similar to those in the left arm. The patellar reflex is greatly exaggerated, and there is distinct ankle-clonus. Motility is impaired, but not completely lost, as he is able to walk with the aid of a cane held in the right hand.

Dr. C. H. Reed has kindly furnished me with the notes of the eye-examinations: At first there were choked disks, conjugate deviation, unequal pupils, and left hemianopsia; these disappeared, but the form fields remained diminished, and the right halves of the retinae less sensitive. The case is interesting because it enables us to determine approximately the sensory constituents of astereognosis.

The stereognostic sense is an exceedingly complex perceptive phenomenon, involving an intricate correlation of tactile-, position-, pressure-, localization-, and temperature-perceptions, and its interpretation by the higher psychic centers; but it does not follow that all these forms of sensation are equally important and that none of them are dispensable. Hoffman² states that the stereognostic may be lost when the temperature-, contact-, pain-, localization-, and muscular-sense are preserved. The recognition of area, pressure, passive movement, and the position of the limbs appears to be more important, although any one of them may be diminished, or even only one preserved, without loss of the stereognostic sense. The most important elements are the recognition of space and of pressure. If motion is impaired, and there is also anesthesia, the results are uncertain. Largely due to Hoffman's teaching, it has been the fashion to ascribe astereognosis in great measure to the loss of muscular sense, although he does not explicitly state that the latter is of such importance. His last statement is difficult to understand. Tactile perception must, of course, be present, for it is inconceivable that a person suffering from total anesthesia should be able to recognize an object by touch. Pressure- and temperature-sense are more concerned in the recognition of the density and nature (by their heat-abstracting power) of objects. The relative parts played by the position- (muscular) and the localization-sense are more difficult to determine. In the case reported, tactile sense is preserved, and the stereognostic sense is, nevertheless, lost. We must, therefore, assume that if the lesion is in, or peripheral to, the perceptive sensory centers, as is evident from the fact that the sense is preserved on the other side of the body, one of the lacking sensations, or a combination of two or more of them, is indispensable to its existence.

The patient had almost complete loss of muscle-sense, inability to distinguish moderate changes of pressure, failure to recognize the consistency of bodies, and disturbance of the localizing sense of the left hand. Every effort was made when an object was placed in his hand, by manipulating the fingers,

² Hoffman, quoted by Möbius, *Diagnostik der Nervenkrankheiten*, Leipzig, 1894.

and changing the position of the object, to obtain some information regarding its nature from him. Occasionally, after moving the object about, he would guess correctly that it was long or short, thick or thin, or make some other general statement that indicated a certain degree of perception. More frequently, however, he would insist that it was impossible for him to recognize any of its features. In this case the fingers remained passive, and muscle-sense was, therefore, largely excluded in the examination, even aside from the fact that it was lost. The following cases have been recorded more or less fully, and aid in throwing light upon the subject.

Wernicke³ records the case of a tailor's apprentice who was struck upon the head with a stick. Immediately afterward there were loss of speech, diminution of power, and considerable inco-ordination of the right arm, and the following day, after the removal of the splinter of bone, complete paralysis of the fingers and hand, although movement of the forearm and arm remained. The muscle-sense, that is, the recognition of passive movement, was completely lost. There was diminution of tactile sense, but no alteration of pain- or temperature-sense. In the course of a few days, there was some improvement in the power of the hand, but a permanent disturbance of the stereognostic sense was discovered, which after five years had not greatly improved. No record is given concerning the ability to localize contact.

von Monakow⁴ reports the case of a man, 25 years of age, who was struck by a piece of wood in the left parietal region. Upon recovering consciousness, the patient noticed that the right arm was paralyzed, and had completely lost sensation. At the operation, a splinter of bone was found in the posterior central convolution. Eight days later motion had returned to a considerable extent, but the thumb and fingers remained parietic. The localization of tactile impressions and the temperature sense were somewhat diminished, the muscle-sense was much disturbed, and there was complete loss of the stereog-

³ Wernicke, *Arbeiten aus der psychiatrischen Klinik in Breslau*. Heft 2, page 235. Leipsic, 1895. Quoted by v. Monakow in *Gehirn-pathologie*.

⁴ v. Monakow, *Gehirnpathologie*, Vienna, 1897.

nostic sense. Several months later, this loss of the stereognostic sense persisted, although it was no longer absolute, but the other sensory disturbances had diminished. It is not noted whether the recognition of passive movements was still lost, but there was some ataxia of the right arm, especially noticeable in writing and drawing.

Burr⁵ reports the case of a man, 24 years of age, who, when 10 years old, received a blow on the side of the head, causing a depressed fracture of the right parietal bone, over the motor area. Several months later after the post-traumatic paralysis and anesthesia had disappeared, the patient discovered that he had completely lost the stereognostic sense in the left hand. At the time of examination, the tactile sense was normal on both sides, but there was complete loss of the localization of contact on the left side. Temperature- and pain-sense were normal, and he was able to recognize passive motion promptly and correctly.

Olmsted⁶ reports the case of a man, 27 years of age, whose sickness commenced with double vision, followed by numbness in the right thumb and fingers. In the right hand, the senses of touch, pain, heat and cold were impaired. Later, there was marked ataxia in the right arm and hand, disturbance of writing, and dulness of the sense of pain. The tactile and temperature-sense were still normal, but there was absolute astereognosis of the right hand. This loss of the recognition of objects apparently extended over the whole of the right side, that is to say, the patient could not recognize a brush, a hard object or the shape of the object on this side, while he was able to do so perfectly on the left side. The pressure-sense was also completely lost. A bottle containing 3 ozs. of mercury appeared as heavy as one containing 9 ozs. Passive movements were recognized at once.

In all these cases, including my own, the tactile sense appears to have been practically normal, or, at least, only slightly diminished. It is therefore possible, to a certain extent, to determine what other elements are capable of causing the loss of

⁵ Burr, *Journ. of Nervous and Mental Disease*, Jan., 1898; also *Univ. Med. Mag.*, Oct., 1897.

⁶ Olmsted, *Journ. of Nervous and Mental Disease*, Nov., 1898.

the stereognostic sense. I have been unable to obtain the original report of Wernicke's case, but in the abstract given by von Monakow, it is not stated whether the sense of localization was lost or not. In von Monakow's case, this sense was diminished, and in Burr's and mine completely lost. Olmsted apparently failed to test it. The muscle-sense was completely lost in Wernicke's, disturbed in von Monakow's, and intact in Burr's and Olmsted's cases. In mine it was lost. Olmsted mentions disturbance of the pressure sense, which was lost in my case. In regard to the other sensations, in Wernicke's case the pain- and temperature-sense were undisturbed; in von Monakow's, the temperature-sense was slightly diminished; in Burr's case, the temperature- and pain-sense were normal; in Olmsted's they were slightly impaired, and in my case temperature-sense was lost, and pain-sense diminished. It appears, therefore, that in all cases in which record was made of the fact, the localization-sense was lost or greatly impaired. The muscle-sense was lost in two cases, normal in two and disturbed in one. The sense of pain was disturbed in two cases, and the temperature-sense lost in one and impaired in another.

In order to determine the relative importance of the muscle-sense—that is, the sense of position—and of the sense of localization of tactile impression, I have conducted, rather roughly, a series of experiments upon normal persons. These were in general performed as follows: the subject was instructed to lay the hand, back downward, flat upon the table, and then, while it remained passive, various objects were placed upon the palm and fingers. If their lower surfaces were irregular, or if the objects themselves were very light, pressure was used to make the contact more distinct. As a result, in nearly all cases the size of the object was correctly given, and even its exact nature was suspected. The perception of size was slightly better on the fingers, in some instances, than upon the palm. The thumb and one of the fingers were then brought in opposition, the object placed between them, and the patient requested to estimate the diameter of the object included in the grasp. This was frequently done with as great accuracy, but never with greater accuracy, than when the object was laid upon the passive hand.

In two cases, one of them a draughtsman, familiar by occupation with dimensions, it was distinctly inaccurate. The fingers were then flexed toward the palm, and the object placed between them and the palm of the hand. Seven subjects expressed a sense of decided difference between the diameter as estimated by this method and as estimated by opposition; the diameter appearing to be very much less, sometimes the difference being as much as half an inch in two persons tested. In an eighth subject, an engineer familiar with measurements, the diameter by this method appeared to be considerably larger, and he expressed the opinion that a cent held thus was a larger coin than a five-cent piece held between the thumb and finger. When the subjects were requested to separate the fingers or a finger and the thumb a certain distance, one of them was able to do so with extraordinary accuracy, the error usually being less than an eighth of an inch. None of the others, perhaps from lack of practice or on account of a less delicate sense of perception of distance, were able to approach this result. It seems from these experiments, that the contact-sense, if I may so call the combination of pressure-, tactile- and localization-sensations, is considerably more accurate than the simple muscular sense, that is, a knowledge of the exact position of the finger or hand, and these results coincide with those obtained by a study of the pathological cases reported.

The stereognostic sense is, however, not merely a matter of sensory perception. The object to be recognized is necessarily complex, and, as I have shown, a variety of sensations are employed in its recognition. It follows that a correlation of these sensations must take place in some higher center or complex of centers. This must then be compared with similar correlations that have taken place in the past, and recognition must be accomplished by the aid of memory. It would seem *a priori* possible, therefore, that cases should occur in which there were no sensory disturbances, and, nevertheless, there was loss of the ability to recognize the nature of objects. Such a case was recently admitted to the Presbyterian Hospital under the care of Dr. Musser.

Case II.—The patient, a music-teacher, 55 years of age, on the 24th of December, 1898, while talking with some friends, suddenly discovered that the left side was paralyzed. There

had been no loss of consciousness, and there were no symptoms of shock. When examined, four days later, the tactile sense on the whole of the left side was greatly impaired. It was completely lost in the forearm and hand and in the leg and foot. Muscle-sense was completely lost in the left arm, although the patient seemed to be able to recognize the position of the arm to a certain extent by its weight. Temperature-, pain-, localization- and pressure-sense were completely lost, although this loss was not absolute above the elbow. On the left leg, similar, but not quite as severe, disturbances of sensation were found. The patellar reflex was present on the left side, but not exaggerated. The Achilles-tendon reflex was lost, and there was no ankle-clonus. Sinkler's reflex, *i. e.* toe-jerk, was present. The stereognostic sense was naturally completely lost on the left side. On the right side all forms of sensation were normal. The stereognostic sense was, however, extremely imperfect on this side. The fields of vision were roughly tested, and the patient found to be suffering from complete left lateral hemianopsia. The following day, Dr. Schaffner, at my request, made an examination, and was able to detect, in addition, a horizontal hemianopsia, so that the patient only perceived the upper right quadrant of the normal field of vision, and even this was considerably contracted. There were no changes in the fundus. On the following day, the sensory phenomena remained unchanged on both sides. The stereognostic sense was, however, normal on the right side. On both sides the power of recognizing writing on the hand was lost. From this time rapid improvement occurred, and three weeks later, the patient was able to give some description of objects placed in the left hand, and in the course of a few days, he made a not altogether unsuccessful attempt to play the organ.

A case somewhat similar to this has been reported by Burr⁷. The patient, a colored woman, 60 years of age, had been suddenly attacked by partial failure of vision, which rapidly increased until she was unable to distinguish objects. She was usually apathetic, but not apparently insane. There was no paralysis, but the patient staggered slightly when walking. Speech was normal. Touch-, pain- and temperature-sensations were everywhere normal, and the point of contact was correctly localized. She was unable to recognize an object placed in the hand, although the loss of the stereognostic sense was not absolute, for it is stated that she could recognize a silver half dollar and the finger. Burr regards the case as one

⁷ Burr, Journal of Nervous and Mental Disease. 1897.

of mind-blindness. He also mentions a similar case that he saw in Weir Mitchell's clinic, in which the patient had had loss of the power of recognizing coins, but this had returned later. In both of these cases, the characteristic feature of the loss of the stereognostic sense, that is to say, the loss on the right side in the case I report, and on both sides in Dr. Burr's case, was the preservation of all the cutaneous sensory perceptions, and it seems justifiable to locate the lesion somewhere in the higher centers by which objects are recognized. This is not necessarily a definite group of cells, for Brissaud⁸ contends that even such well-organized actions as writing or reading are performed, not by definite centers, but by a group of centers, the communications between which have been rendered more direct by constant employment.

Exner⁹ and Ziehen¹⁰ have also suggested a similar theory for apperception, basing it upon the well-known physiological law, that the irritation of a portion of the central nervous system leaves, for a short time, an increased irritability in the excited part. Indeed, Exner suggests that memory is largely the result of the increased irritability of certain complex routes.

Kattwinkel¹¹ has recently investigated a somewhat analogous subject—the ability of the patient to recognize letters written with a blunt object upon the skin. He finds that this is lost in conditions in which there is alexia or sensory anesthesia. Even in the former condition, mere sensation may be so accurate that the stereognostic sense is preserved; as occurred in a case of a man 74 years of age, who suddenly lost his speech as the result of an accident. There was, however, no hemiparesis and no disturbance of sensation. Kattwinkel suggests the theory that this ability to recognize letters is due to the combined action of the tactile, visual and speech-centers, and it is highly probable that a similar combination of mental processes is necessary for the recognition of objects.

The following conclusions appear to be justified:

1st. The stereognostic sense is due to a complex sensory

⁸ Brissaud, *La Presse médicale*, 1898.

⁹ Exner, *Physiologische Erklärung der psychischen Erscheinungen*. Leipzig, 1894.

¹⁰ Ziehen, *Leitfaden der physiologischen Psychologie*, June, 1893.

¹¹ Kattwinkel, *Deutsches Archiv für klin. Med.*, Oct. 27, 1898.

perception, the impulses of which are probably received in the parietal lobe of the brain, and then recognized in consciousness by some higher center or group of centers. It may be lost if either one of these centers is disturbed.

2d. In case of peripheral disturbance, that is, some disturbance in or between the center in the parietal lobe and the terminations of the sensory nerves in the skin, stereognosis is lost if tactile sense is lost, and it is greatly impaired or lost if localization-sense is lost; it is usually, but not always, disturbed if the muscular sense is lost, but may persist if any of the other forms of cutaneous sensation are lost or disturbed.

3d. The apperceptive center may be disturbed as a result of severe injury to the brain, as, for example, in apoplexy, and in these cases the stereognostic sense may be lost, even although all forms of sensory phenomena are preserved.

I desire to express my thanks to Dr. John H. Musser, who has generously permitted me to use the two cases here reported.

31. STATISTISCHE BETRACHTUNG ÜBER ALLGEMEINE PARALYSE, NACH DEM MATERIAL DER OBERBAYRISCHEN KREISIRKENANSTALT GABERSEE (Statistical Contribution on General Paresis from Gabersee), v. Oscar Müller (*Allgemeine Zeitschrift f. Psychiatrie*, 54. 1898, p. 1027).

At the Gabersee institution during the years 1883 to 1897 inclusive, there were admitted some 1,158 patients, of which 122 or 10.6 per cent. suffered from general paresis. This number served as a basis for the author's statistical contribution. A large number of these patients were women; 45 in number, the men being 77; or 1 woman to every 1.7 men, which is an unusually high average. Among the etiological factors, syphilis seemed low, in the men being found in 17 per cent., and in the women 6.4 per cent. Syphilis or alcohol together gave a general average of some 60 per cent. Heredity, especially nervous disease in the parents, seemed to play an important role. It was found in 46 per cent. of the men and 64 per cent. of the women. The average age of onset for men was between thirty-six and forty, while for women two periods seemed favorable, from twenty-six to thirty, and from forty-five to fifty. The duration varied in the men from seven months to seven years, in the women from one year to nine years. Thirty-six cases died in convulsions. There were over eighty post mortem examinations recorded. Among the statistics given with reference to the findings, it was shown that at least 50 per cent. showed chronic pachymeningitis externa, 81 per cent. showed distinct granulation of the ependyma, 76 per cent. had enlarged ventricles, and in 96 per cent. the brain showed the characteristic gelatinous serous infiltration. In four cases only were there any softenings found, and only one case of new growth was observed.

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THE NEW YORK NEUROLOGICAL SOCIETY.

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The President, Dr. Frederick Peterson, in the chair.

A CASE OF DISSEMINATED SCLEROSIS.

Dr. L. Stieglitz presented a boy, seventeen years of age, one of a family of nine children. None of the others showed a similar condition. When the patient was six years of age, he was noticed to walk peculiarly, and a year later he began to have attacks of intense headache and vomiting. About three years ago the headaches became less severe, but recently they have had their former intensity. He often has trouble in urinating. Examination shows a fairly well developed boy, presenting the typical gait of cerebellar ataxia. The muscular power is generally diminished, particularly in the right arm. There is a distinct hemiplegia, nystagmus in both eyes, and divergent squint. The fundi appear entirely normal. The speech is thick, nasal and drawling.

The speaker said, regarding the diagnosis, that if the affection had been more recent, he would have been led to think of brain tumor. Syphilis of the brain could be excluded, not only on account of the personal and family history, but because of the negative results under antisyphilitic treatment. Friedrich's ataxia could be excluded because of the hemiplegia and increased knee-jerks. He looked upon the case as one of disseminated sclerosis. Plaques in the cerebellum would account for the cerebellar ataxia, and plaques in the left hemisphere would account for the right hemiplegia.

Dr. Graeme M. Hammond was not quite ready to accept the diagnosis of disseminated sclerosis. There were changes in the mentality, and a younger sister exhibited evidence of beginning degeneration of the brain. He was inclined to think that there was atrophy of all parts of the cerebrum.

Dr. Steiglitz did not think that the deficient mentality negated the view that the case was one of disseminated sclerosis. He had not seen the other children.

INSTRUMENT FOR MEASURING THE PATELLAR REFLEX.

Dr. W. H. Haynes exhibited an instrument which he had devised for measuring the reflexes, and demonstrated its action. The apparatus consists of an oblong iron stand on castors. To

one end of this is attached an upright iron rod carrying a large metallic semicircle with convenient graduations. At the center of the upright rod an aluminum arm is hinged, and the other end of this arm swings along the brass arc and can be fastened by a screw-clamp. The aluminum arm is placed against the tip of the patient's foot, and the reflex tested. The arm is kicked upward by the foot to a greater or less extent, and the spring clamp is left clinging to the point on the arc to which it was kicked.

Dr. L. Muskens said that he had been working with another instrument intended for the same purpose. His work in this line had taught him that it was not so much the liveliness of the knee-jerk as its quality which was of importance, both in research and clinical work. The readings in such an apparatus were also greatly vitiated by the adjustment of the instrument and the angle at which the foot strikes the arm.

Dr. Charles K. Mills, of Philadelphia, read a paper with the title: "Some points of special interest in the study of the deep reflexes of the lower extremities." (See page 131.)

Dr. Charles L. Dana said that he did not recall having met with ankle-clonus in connection with absence of the knee-jerk. He was absolutely in accord with Dr. Mills and Sir Wm. Gowers as to the connection of ankle-clonus with hysteria. He did not believe that in true hysterical paralyses there is any ankle-clonus; if it does occur, it is due to some toxic or organic disorder. At one time he had studied a series of 20 cases of hysterical paralyses, and had been impressed by the fact that the knee-jerks were diminished and the tonus somewhat lessened in this form of paralysis. But there is a form of paralysis which might be considered, in which there is an ankle-clonus. He had recently seen a lady of forty years who had had for a number of years certain neurasthenic symptoms, and who finally suffered intense pain in the back, and developed partial paraplegia. There was at this time a genuine and persistent ankle-clonus. After about one month the patient recovered completely. He had since seen a similar case in which were spinal irritation, neurasthenic symptoms and functional paraplegia. These cases, he thought, were not true examples of the hysterical state, but of profound nerve exhaustion. These exceptions do not vitiate the general statement, that hysterical palsies are not accompanied by ankle-clonus.

Dr. B. Sachs said that he had had under observation, some years ago, a patient with absolute loss of knee-jerks, but presence of ankle-clonus, in whom the preservation of ankle-clonus was in all probability due to the fact that the lesion was limited to the upper lumbar segment. He had also witnessed the same peculiar association of symptoms in a case of Pott's disease in which the lower thoracic vertebræ were involved. He believed that in this case, too, the lower lumbar and sacral segments had entirely escaped. He was disposed to agree entirely with the view taken by Gowers and Mills regarding the relation of ankle-clonus to hysteria. He had noted ankle-clonus in a case of distinct hysterical paraplegia in a child, and had observed the same thing once in a young man suffering from neurasthenia. It is a very curious fact, showing the relative value of the deep reflexes, that while the knee-jerks are so commonly exaggerated in neurasthenia, ankle-clonus is almost invariably wanting.

As to the question of the dependence of the absence of knee-jerk upon the degeneration of the muscle fiber, it should be noted that this was probably the explanation of the absence of the knee-jerk in muscular dystrophies.

Dr. G. M. Hammond said that he agreed with the last speaker in regard to the absence of ankle-clonus in hysteria, as a rule, and yet he had seen at least three or four cases of hysteria in which ankle-clonus had certainly been present. When such conditions exist it is exceedingly difficult to make the differential diagnosis between hysteria and organic disease. For some years he had been making observations in cases of this kind concerning the effect of etherization. The ankle-clonus was found to be maintained, even under full etherization, when there was an underlying organic disease, although this was not the case in hysterical subjects.

Dr. W. M. Leszynsky said that during the past summer he had had under observation a man with certain symptoms of transverse myelitis. Examination showed the absence of both knee-jerks and presence of ankle-clonus on both sides. In addition, there was a clonus affecting the gluteus maximus muscle on one side. He had seen ankle-clonus at least three times in patients who had been under observation for a number of years, and who had not developed any organic disease. He asked Dr. Mills if he had ever noticed ankle-clonus produced by tapping the Achilles-tendon while the foot is flexed in the usual manner. It was only in this way that he had been able to demonstrate it in two or three instances.

Dr. Joseph Collins asked Dr. Mills whether he believed that the actively contractile substance of the muscle in his first reported case was in such a diseased state that it was thrown into clonus by the tap on the tendon. Regarding the occurrence of clonus in hysterical conditions he said that he had seen cases of traumatic hysteria, in which there was a true ankle-clonus, and in which recovery had taken place. He could not differentiate such cases from true hysteria.

Dr. F. Peterson said regarding the presence of ankle-clonus in hysteria, that he had never seen a genuine clonus in a case of pure hysteria.

Dr. Mills closed the discussion. In answer to Dr. Collins he said that the explanation in the case he had reported was not very clear, but he believed that the loss of knee-jerk was due to a combination of muscle and neural disease, both of which were demonstrated by the microscope. Probably the nerve to the tendo Achillis had been in a state of neuritis earlier, and had so caused an excitation of the ankle phenomenon. In the examination of some of the cases of tabes for tendo Achillis jerk, slight clonus was found in two. He had elicited tendo Achillis clonus by tapping on the tendon when ankle-clonus could not be elicited by simply placing the foot in dorsal flexion. The patient's knees should be placed close to the chair on which he leans, and the tendon should be percussed near the heel. As to the question of ankle-clonus in hysteria he would again emphasize the necessity for more explicit statements regarding it. In every case one should discriminate carefully between spurious, abortive clonus and the clonus of the uniform and persistent type. He took the position that in a very limited number, probably less than five per cent., the abortive or spurious type of ankle-clonus would be found. In one definite and limited class of cases usually regarded as hysterical, and sometimes with other stigmata of hysteria, such as the sensory changes, one meets with what appears to be ankle-clonus. This is the class of cases characterized by hypertonicity. True ankle-clonus with hemiparesis or hemiplegia he considered almost pathognomonic of organic disease.

A CONTRIBUTION TO THE STUDY OF CEREBRO-SPINAL
MENINGITIS.

Dr. Lewis A. Conner read a paper with this title. The paper was based upon 60 such cases, occurring at the Hudson Street Hospital and New York Hospital. Every effort had been made to exclude those in which the meningitis was secondary to other processes. Of the 60, 21 occurred in 1893, the year in which the disease was especially prevalent in various parts of the country. Ten occurred in each of the last two years. One-half of all the cases occurred in the spring months, March, April and May. Seventy per cent. occurred in males; 32 per cent. in those under the age of fifteen years, 43 in those between fifteen and thirty, and 25 in those over thirty years. The youngest case was that of a child of two months, and the oldest a woman of sixty-five years. In only one was there a distinct history of exposure to cold immediately preceding the attack. No two of the cases came from the same family, although two negroes occupying the same room were attacked within a few hours of each other, and both died in a short time.

Post-mortem examinations were made in 18 of the 41 fatal cases. In the very acute cases there was marked congestion of the meningeal vessels. The exudate was often so slight as to be scarcely perceptible, except in cases that had lasted two weeks or more. The lateral ventricles were often distended with purulent fluid. The greatest involvement of the cord was in the thoracic and lumbar portions, and always on the posterior surface. Microscopical examination showed constant involvement of the subjacent brain tissue. The common involvement of the cranial nerves was also noted.

Councilman, and his associates, are convinced that the specific cause of epidemic cerebro-spinal meningitis is the diplococcus intracellularis. In the 35 cases examined by them the organism was found in all but four. Bacteriological examination was made in ten of the cases reported in the present paper. In three autopsies in which the culture results were negative only ordinary culture media were used. The diplococcus intracellularis was found in only four of the cases, and the pneumococcus in about the same proportion.

All of the usual types of cases described were represented in this series. All of the fulminant cases were marked by early delirium and coma. Twenty-six ran their course in two weeks, and all ended fatally. There were two of the intermittent type. Thirteen lasted more than one month, and of these, 11 recovered, the longest being for three and a half months. The onset was sudden in 39 cases. The temperature was markedly irregular, but there were several distinct types. In a number the

temperature was normal or subnormal throughout the disease, or up to a short time before death. In a second type the temperature was moderate most of the time. In others, the temperature rose gradually, and then fell whether or not recovery took place. These cases resembled somewhat typhoid fever, but not infrequently the highest temperature was in the morning. In the fulminant cases the temperature was sometimes nearly normal for the first few hours. The pulse also varied greatly, and was independent of the variations in temperature. Retardation of the pulse was not common or marked except in two cases. When the symptoms were active, an almost constant symptom was a marked increase in the respiration-rate. Vomiting was present at some time in over one half; in 25 it appeared at the onset, and in 15 only at that time. In 15 it remained a prominent feature throughout the disease. In no case was the vomiting "projectile." There was more or less pain in all cases, and it was usually in the form of an intense occipital headache, but pain in the back, limbs and chest was not infrequent, and sometimes was especially marked on one side. The reflexes presented no constant feature. Greater or less rigidity of the muscles of the head, neck and back was present in almost every case. Rigidity of all the extremities was also common. There was no uniformity about the appearance of the pupils. In one case which recovered consciousness was preserved throughout the disease. In a large proportion of the cases delirium and stupor alternated. Some of them were marked by an unusual form of delirium, simulating hysteria. Skin manifestations were observed in 25 per cent., but the hemorrhagic eruption was seen in only three of the cases. A distinct leucocytosis was present in most of the cases in which the blood was examined. The spleen was enlarged in 23 per cent. of the cases. In none of the patients who recovered was vision disturbed subsequently. Two patients had bilateral deafness, and one of these recovered completely. In several there were involvements of the joints resembling those seen in rheumatism. In seven pneumonia occurred, and in all it developed after the meningitis. Albuminuria was generally an accompaniment of the disease.

Dr. Joseph Collins said that he thought the paper went to prove, rather than disprove, the theory that attacks of cerebro-spinal meningitis are entirely distinct in their causation and pathogeny from those which develop sporadically. In very few of the latter cases was the *diplococcus intracellularis* found, whereas it was almost constantly present in the epidemic cases studied by Councilman. The cases reported in the paper had been clearly shown to be of the non-epidemic variety. He would, therefore, still maintain the opinion published elsewhere, that the epidemic form is entirely different in its causation and pathogeny from the sporadic, the former being associated only with an individual organism. It was true; however, that the

latter was commonly associated with the diplococcus of pneumonia.

Dr. Sachs thought the statistics just presented in the paper might lead to some confusion. He did not think the diagnosis of epidemic cerebro-spinal meningitis could be made positively at the present time without post-mortem examination. The prognosis in this series was evidently rather better than that usually observed in epidemic cerebro-spinal meningitis. Recently he had seen a girl of eighteen who within a few hours was comatose, and died within twenty-four hours. The autopsy revealed the diplococcus intracellularis. Such cases should hardly be placed in the same category with the epidemic form.

Dr. Conner said that he had refrained from using the term "epidemic meningitis," because he had been unable to convince himself that epidemic and sporadic meningitis were different diseases. One or two of the cases in which the pneumococcus was found occurred in 1893, a year in which this disease was epidemic. A case which developed last summer after exposure to the sun, was observed for two months, and was examined after death, and the diplococcus intracellularis was discovered. In this case, as well as in the one referred to by Dr. Sachs, the disease was evidently sporadic, and yet the diplococcus intracellularis was present. He could not therefore accept the statements made by Dr. Collins. It seemed to be impossible to distinguish clinically between the two forms. The mortality in his cases was 65 per cent., exactly the rate found by Councilman in the epidemic at Boston.

32. UEBER EINEN FALL VON JUVENILER PARALYSE AUF HEREDITAER LUETISCHER BASIS MIT SPECIFISCHEN GEFÄSSVERÄNDERUNGEN. A Case of early Paresis due to Congenital Syphilis with specific changes in the Blood Vessels. C. v. Rad. Archiv für Psychiatrie und Nervenkrankheiten, 30, 1898, p. 82.

A young man who had previous been healthy, but whose father was syphilitic, at the age of 15 developed attacks of convulsions combined with periods of unconsciousness, an increasing mental weakness having begun to manifest itself for some time before. In addition to these epileptiform attacks marked decline of the visual and mental powers set in, and at the age of 21 had reduced the patient to a condition of absolute idiocy. Other objective symptoms were also observed, such as pupillary non-reaction, spastic paraplegia of the legs and disorders of speech. After 7 weeks in the hospital the patient died from an attack of pneumonia.

The autopsy fully explained the clinical picture. The thickening of the pia, degeneration of the large pyramidal cells and disappearance of tangential fibers was clearly indicative of paresis, while the capillaries of the cortex were thickened and presented localized dilatations and in places were surrounded by bloody extravasations of various ages. In the cord there was found a moderate degree of degeneration of the pyramidal tracts with the balance of the white matter normal, as not infrequently is the case in paralysis. The basal vessels were in a condition of syphilitic endarteritis, the intima being everywhere thickened and in the smaller vessels a peri and mesarteritis were found. The author emphasizes the specifically syphilitic changes of the basal vessels as they plainly demonstrate the surmised, but not yet definitely proven, connection between congenital syphilis and paresis in the young.

THE PHILADELPHIA NEUROLOGICAL SOCIETY.

November 28, 1898.

The President, Dr. F. X. Dercum, in the chair.

Dr. H. C. Wood reported three cases in which pain was an important symptom, and in two of the cases was accompanied by muscular atrophy and paresis. The paper will be published later in this journal.

Dr. Charles K. Mills said that he had had the opportunity to examine the third case with Dr. Wood. He was impressed with the fact that although these three cases occurred in the same family, yet it was not improbable that they did not represent a family type. It was almost impossible to make a diagnosis from the history of the first case, but the record did not seem to permit of it being placed with the other two. While the diagnosis in the third case was difficult, he thought that Dr. Wood's opinion was as nearly correct as any that could be formed. In regard to the second case, he was unable to come to a conclusion. In some of its features it looked like a case of anterior poliomyelitis with pain. The history was peculiar and unlike that of the other two cases.

Dr. William G. Spiller agreed with Dr. Mills that these cases need not be regarded as examples of the same disease. He could not form any idea of the nature of the first case, as the history was incomplete.

The second case, he thought, was probably due to disease of the peripheral neurons. The man had muscular atrophy, reaction of degeneration, loss of muscular power, loss of the patellar reflex in the affected limb, and pain; these are symptoms which point to disease of the peripheral sensory and motor neurons.

The diagnosis in the third case was much more difficult. It might possibly be a very atypical case of amyotrophic lateral sclerosis. The posterior root entrance zones could hardly be much altered. It would be difficult to understand how the reflexes could be exaggerated if these zones were much involved. The reflexes cannot be increased if the sensory portion of the peripheral arc is destroyed, even though the central motor fibers are entirely degenerated. Some of the features in this third case were very unlike those of amyotrophic lateral sclerosis.

Dr. H. C. Wood said that he did not assert that these three cases represented the same disease, but where things are similar it seemed probable that they were due to the same cause. These cases had the three prominent symptoms of pain without tenderness, increasing loss of motility and wasting of the muscles. There must therefore be a suspicion that they were due to the same cause.

He did not believe that simple amyotrophic lateral sclerosis is associated with pain as severe as that which occurred in the third case; he therefore believed that there must be some other lesion present.

A CASE OF PERIODIC FAMILY PARALYSIS.

Dr. J. K. Mitchell presented a patient, the subject of periodic family paralysis. The patient's mother, an uncle, a first cousin, a grandfather and a first cousin of the grandfather, all on the

maternal side, had suffered with varying degrees of the same disorder. The patient, aged 18, had his first attack five years ago, since which time they have steadily increased in both frequency and severity, paralysis now occurring about once in eight days, and usually lasting from 36 to 48 hours. He wakes from sleep, unable to move, the disability at first being imperfect, but increasing through the day. During the height of the seizure he can move his head from side to side, but not any limb. All reflexes are lost in severe attacks; plantar, knee, cremaster and abdominal. In the lighter degrees of the palsy the reflexes are slow and small, and very slightly reinforcible. Electric reactions are totally absent in the muscles of the lower limbs in severe, and only producible by very strong galvanic currents in slight attacks. There is during the attack a rough distinct murmur with a splitting of the second sound, heard at the third left costal cartilage, and less clearly along the left sternal margin. The pulse during palsy is sometimes slower than the patient's normal pulse, sometimes faster, and with a good deal of tension. Voice, breathing, swallowing, are unaffected, and the mental faculties perfectly unimpaired. It is sufficient to say without too much detail, that in the intervals the patient is in absolutely perfect health and normal in all respects. Urine, feces and blood have been examined with as yet little result, but this investigation is still in process.

The patient being brought before the society said that since coming to the room he felt the usual warning of an attack in the form of slight general weakness. On attempting to develop his reflexes the knee-jerk was found to be markedly slow and small.

Dr. Charles K. Mills remarked that it seemed impossible to explain such a case as this except on the theory of a temporary toxemia.

Dr. E. Lindon Mellus, of Baltimore, said that the history of this patient suggested to him some of the cases of post-epileptic paresis without epileptic paroxysms.

Dr. E. Lindon Mellus, of Baltimore, read a paper on "Motor Paths in the Brain and Cord of the Monkey." (To be published in this journal.)

Dr. William G. Spiller stated that he wished to report the discovery of a tract not previously described. Some time ago, Dr. Hare reported to this society a case of edema due to a hemorrhage in the external capsule and the lenticular nucleus. The brain was given to Dr. Spiller for examination. In this specimen the latter had found degenerated fibers that certainly came from a level as high as the upper part of the thalamus. They passed down with the degenerated motor tract through the crus, and entered the pons closely associated with the motor tract. A little below the level of the exit of the fifth nerve, a band of fibers became separated from the outermost and lateral portion of the pyramidal tract. Lower down in the pons this band of fibers passed quite abruptly backward and entered the trap-

ezoid body. It took a position at the level of the junction of the medulla oblongata and pons, lateral to the uppermost portion of the inferior olive. As the inferior olive increased in size, this bundle passed backward, and where the olive had its largest width the bundle took a position on the posterior and lateral side of the lower olive. Dr. Spiller had traced this bundle below the motor decussation and had found that it remained on the same side throughout its entire length. He had followed this band of fibers from the level of the upper part of the lenticular nucleus as far as the first cervical segment. In this specimen he had also traced motor fibers to the lateral tract on the same side of the cord, as Dr. Mellus had done in the monkey.

Dr. Spiller agreed with Dr. Mellus in the statement that the failure to stain the degenerated collaterals of the pyramidal fibers within the anterior horns might be due to the fact that these fibers are so small that the degenerated myelin is not very distinct; he did know, however, that the medullary sheath remains on the reflex collaterals of the posterior roots after these collaterals enter the gray matter. Dr. Dejerine and he had been able to trace collaterals of the posterior roots into the anterior horns.

The black dots which Dr. Mellus had shown in the perivascular spaces and had regarded as contained within the cells lining these spaces, Dr. Spiller had seen a number of times, and believed that they were contained within the granular bodies, the "*corps granuleux*" of the French writers.

December 19, 1898.

Dr. Charles K. Mills in the chair.

A CASE OF CEREBELLAR TUMOR.

Dr. Spiller presented a woman of 43 years who began to have severe headache four years ago. The headache lasted two years, then disappeared, and during the last few weeks had returned over the left side of the head behind and above the ear. This region was very sensitive to the slightest touch. The woman had had extreme vertigo during the past two years. She could lie on her right side, *but lying on her left side caused unendurable vertigo*. She had not had vomiting, disturbance of the sphincters, or pain in the limbs. The gait was extremely cerebellar in type and Romberg's sign was pronounced. The patellar reflex was absent on each side, and tactile sensation below the knees was diminished. A tendency to fall backward was very evident. The reaction of the pupils was normal, and the cranial nerves were not affected. The optic disks were somewhat congested. A history of a discharge of pus from the left ear two years ago was given, and Dr. Randall found healed perforation of each ear. No word-deafness, no word-blindness, no hemianopsia, and no mental disturbance existed.

Dr. Spiller regarded this case as one of cerebellar disease, probably tumor or abscess situated in, or pressing upon, the cerebellum; although dilatation of the fourth ventricle or meningitis might cause such a symptom-complex. The only localiz-

ing symptoms were the extreme vertigo when the patient laid upon her left side, and the great sensitiveness of the region behind and above the left ear. It was extremely difficult, therefore, to suggest to the surgeon the proper place for operation. The sensitive area was indicative of a lesion in the lower part of the left occipito-temporal lobe; the extreme vertigo when the patient laid upon her left side was suggestive of a tumor in the right cerebellar hemisphere, causing pressure upon the cerebellum when the right side of the body was uppermost.

Dr. Charles K. Mills thought that this case was one either of tumor or abscess of the cerebellum, with the probabilities in favor of tumor. The pain was not over the cerebellum but over the occipital lobe, and in the neighborhood of the parieto-occipital fissure. The patient, however, did not have the distinctive symptoms of tumor or abscess involving the left occipital lobe. There was no word-blindness and no hemianopsia. The position of the pain would seem to indicate that the tumor was separated anatomically from the cerebellum. He thought possibly that some pachymeningitis might be associated with the cerebellar tumor. As the case was a serious one and progressing, he believed that an exploratory operation was advisable.

In the study of such cases as this, Dr. Mills said it was of interest to study not only the quadriceps-jerk and knee-jerk, but also the gastrocnemius-jerk and ankle-jerk. In the present case, the muscle and tendon phenomena in the lower extremities were entirely absent. He had recently studied the gastrocnemius-jerk and ankle-jerk in a number of cases of tabes. About twenty-eight cases had been examined, and in all but three the tendo-Achillis jerk was absent. In one case it was present only on one side. In one case it was rather plus. The gastrocnemius-jerk was present in all. This matter might be of some importance in the differential diagnosis of a case of the kind presented by Dr. Spiller in its early stage from a case of tabes. In this patient with cerebellar tumor there was loss not only of the patellar reflex and Achilles-jerk, but also of the gastrocnemius-jerk and quadriceps-jerk. In almost every case of tabes which he had examined in this connection, the quadriceps-jerk and gastrocnemius-jerk were preserved.

Dr. Spiller remarked that the question of the absence of the reflexes in brain tumor is one of great interest and one that we understand very imperfectly. In a certain number of these cases examined post mortem, degeneration of the posterior roots had been found. Numerous papers have been written to show why this occurs. It is held by some that the cachexia causes the degeneration of the posterior roots. By others the theory is held that this posterior root degeneration is due to pressure; that there is an increase of the cerebrospinal fluid and that the pressure is exerted especially in the lower part of the cord. Those who oppose this theory bring forward the statement that the degeneration may involve the cervical cord more than the lumbar, and contend that the pressure theory is not satisfactory.

FIVE CASES OF TABES IN THE NEGRO.

Dr. J. W. McConnell stated that about six years ago in concluding a paper read before this society, Dr. Burr had said that "while the data given are not sufficient to permit one to say dogmatically that locomotor ataxia is a rare disease among

negroes, still they are sufficient to permit one to hold such an opinion tentatively until further investigation disproves it." It was not Dr. McConnell's intention to gainsay the opinion expressed by Dr. Burr at that time, but rather to emphasize it and to add these cases to those now known.

Eight years' service at the dispensary for nervous diseases at the Philadelphia Polyclinic had given him an opportunity to see many cases of tabes in the white race, quite a few among the mixed races, and but five instances of the disease in pure negroes.

Case I.—B., full blooded negro, age 55 yrs., native of Virginia, a sailor by occupation. Earliest symptoms appeared fifteen years ago; sharp, cutting pains in legs and thighs, failure of sight in left eye, two years later right eye became involved. Condition grew progressively worse. Specific history acknowledged. Examination showed no ataxia, no Romberg sign, no paralysis or paresis. Total blindness, pupils contracted and immovable to light stimulation. Ophthalmologist reported a condition of primary optic atrophy in both eyes. Knee-jerks retained, but diminished.

Case II.—G., full blooded negro, parents born in Liberia, age 49 yrs. Twelve years ago had a chancre followed by secondaries. Weakness in the legs for ten years, unsteady on his feet, shooting pains in both lower extremities. Has had girdle sensation, also a feeling as of a veil over his face. At present he is totally blind in the right eye, partially so in left. Typical primary degeneration of both optic nerves. Knee-jerks absolutely lost. He has sharp pains in legs, numbness of the right hand and of the ulnar edge of the left hand. No rectal or vesical trouble.

Case III.—L., negro waiter, born in Virginia, aged 44 years; specific history. Pain in legs for some years, with some difficulty in walking. Lately failing sight. Knee-jerks lost; slight sway, very slightly ataxic gait. Pupils small, disks show advancing gray degeneration.

Case IV.—W., aged 51 yrs., negro born in North Carolina; specific infection, twenty-four years ago, secondaries quite free. Pain in legs commenced fifteen years ago. Diplopia about ten years ago, and soon after failure of vision. At present the patient is almost blind in both eyes, has very small pupils. He is not ataxic, does not sway abnormally. Knee-jerks are abolished. He has difficulty in holding his water and also in controlling the sphincter ani.

Case V.—T., female, full blooded negress, aged 34 (?) years, native of Virginia, married. Possible specific infection ten years ago. Sight has been failing for two years in the right eye, for one year in the left. At present, ataxia of lower extremities, sway increased, sharp pains in legs, girdle sensation, feeling as of a cobweb over her face, difficulty in holding water. Knee-jerks absolutely lost. Is almost blind in both eyes and ophthalmologist reports complete spinal atrophy of both optic nerves. Absolute iridoplegia.

Some features of the cases were worth emphasis.

Four of the patients were men and one a woman. All five were pure negroes. The parents of four were known by the respective patients to have been negroes, and one of these four was the son of parents born in Liberia. The fifth had been a slave, the son of a negro slave father. He did not recollect ever

having seen his mother, but as the man was very black Dr. McConnell presumed that his mother was a negress.

The cases were all of the amaurotic form of tabes. In three instances the failure of vision was gradual and came on after the pains, and affected both eyes simultaneously and in like degree. In the other two the vision of one eye failed before that of the other. Four were almost blind, although one patient was relatively less so than the others. The fifth was reported by the ophthalmologist as "one of pallid disks which were very suggestive of advancing tabetic atrophy." Three of the patients were distinctly ataxic in the lower extremities; the woman had also some difficulty in using the upper extremities. In these three the Romberg sign was obtainable.

The woman and one of the men presented many of the usual text-book symptoms of tabes dorsalis. Two of the other three suffered with lancinating pains, the knee-jerks were abolished, the irides were not responsive to light, the optic nerve was the seat of tabetic atrophy. One of the two had Romberg's symptom. A fifth patient, most blind of all, had shooting pains and girdle sensation, but no ataxia and no Romberg's sign. He had retained the knee-jerks. These were perhaps not as prompt as in a normal individual, but they were present. In all five there was iridoplegia. Two cases had some involvement of the vesical sphincter; two complained of a feeling as if the face was covered with cobwebs or a veil. One case presented numbness of the ulnar edge of the left hand and of the whole of the right hand.

The four men admitted specific infection, and in the woman it was very probable from her own statements.

Dr. William G. Spiller said that three of the cases had come to the Polyclinic Hospital since he had been connected with the service. They were all of the amaurotic type. If the negro is more liable to this form of tabes, it may explain why the disease is considered uncommon in the negro, inasmuch as the amaurotic form is at times overlooked by the general practitioner. A year ago, he presented one or two patients with the amaurotic form, and at the time spoke of Edinger's substitution theory, and said that this theory did not explain the amaurotic form. If the disease occurred in those who used their eyes to excess we might understand why overwork has something to do with it. He had recently received a letter from Prof. Edinger in which the latter, in the spirit of a true scientist, acknowledged that his theory would not explain every condition in tabes, but stated that it explained much better than any other so many symptoms that it must contain a "fragment of truth." Prof. Edinger also spoke of the great importance of the statements regarding the resistance of the negro to tabes. Dr. Spiller referred to the exceedingly interesting experiments which Prof. Edinger had performed on rats.

Dr. John K. Mitchell said that Dr. Burr seemed to assume the almost necessary presence of syphilis in these cases. While he recognized the frequency with which syphilis precedes tabes, yet the more he saw, the more he doubted the supposed necessary relation between

the two. A careful lookout for tabes in the negro had been kept at the Orthopedic Hospital for twelve or thirteen years, and not more than three cases had been found; one of these was in a very light mulatto.

He thought that the blindness might result in a certain concealment of some of the tabetic symptoms: the blind patient, if he can walk at all, necessarily does so without seeing his feet; thus perforce he acquires some of the greater ease of motion and improvement in station which tabetics acquire by learning to execute careful co-ordinate movements—not that they have changed the diseased tissues of the cord, but that they use themselves enough better to conceal some of the symptoms of the disorder.

Dr. Burr replied that he did not believe that syphilis is the only cause of tabes dorsalis.

Dr. Joseph Sailer reported a case of hemiplegia with astereognosis.

Dr. J. T. Rugh reported a case of hysteria simulating ascending neuritis. (To be published later in this journal.)

Dr. F. Savary Pearce said he believed this patient had neuritis with hysteria.

Dr. Spiller had seen this patient in consultation and had found a very serious condition of the left arm. The arm was much swollen. The patient felt tactile sensation at every part of the arm; when a pin was thrust into the skin she felt it as touch and not as pain; she had, therefore, dissociation of sensation. Knowing the great rarity of ascending neuritis, especially in the absence of pus formation, Dr. Spiller was exceedingly skeptical as to its presence in this case. The electrical reactions were found to be perfectly normal, except that a stronger current was required on account of the swelling. Within twenty-four hours after Dr. Spiller first saw the patient the paralysis had become complete and sensation was lost from the shoulder down. The pain, which had been intense, had entirely disappeared. He was convinced that hysteria was the cause of this apparent neuritis. A purely suggestive treatment was adopted and in three or four days all the signs of the supposed neuritis had disappeared.

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33. UN CAS DE PARALYSIE GÉNÉRALE JUVENILE AVEC SYPHILIS HÉRÉDITAIRE (A Case of Juvenile General Paresis, with Hereditary Syphilis). A. Saporito (Ann. méd. Psychol., 6, 1898).

General paresis developed in this young man at the age of 18. There was no particular neuropathic nor psychopathic heredity, but there were evident signs in the patient of hereditary syphilis. He was poorly developed, had an infantile head with Hutchinson teeth and other characteristic marks. He was always somewhat peculiar, but was comparatively bright up to the age of 16, when a gradual dementia began with general elevation of the ego. There were typical changes in his speech, fibrillary tremors of face and tongue, Argyll Robertson pupils, with inequality, epileptoid attacks, marasmus and death. The pathological findings were those usually described as resulting from general paresis.

Periscope.

ANATOMY AND PHYSIOLOGY.

34. CONSIDERAZIONI SULLA STRUTTURA DEL NUCLEO DELLE CELLULE NERVOSI (Considerations of the Nuclear Structure in Nerve Cells). G. Levi (Rivista di Patologia nervosa e mentale, 3, 1898, p. 289).

After a brief summary of the opinions of various authors on the micro-chemical reactions of the nuclear structures, the author gives the results of his own researches. He believes that in the nucleus are found at least two distinct substances, the central portion consisting of a substance stained by acid reagents, the peripheral portions taking up basic coloring matters. In various pathological conditions the peripheral portions remain unaltered, while the central portion of the nucleus may show diffuse acidophile coloration. That the nucleus is not made up entirely of nucleins would seem to be proven by the author's researches. The acidophile substances change greatly during the cellular activities, while the basophile substances, nucleins, remain more or less stable. In the mitotic changes, elsewhere studied more in detail by the same author, the acidophile structures seem to give rise in part to the centrosomes and in part to the spindles.

JELLIFFE.

35. UEBER DIE URSACHEN, WELCHE DIE WACHSTHUMSRICHTUNG DER NERVENFASERN BEI DER REGENERATION BESTIMMEN (The Causes That Determine the Direction of Growth of the Peripheral Nerve Fibers During Regeneration). J. Forssman (Ziegler's Beiträge, 24, 1898, p. 56).

As hitherto a mechanical theory was the only one generally accepted as explaining the direction of growth of nerve fibers, and yet is for many reasons unsatisfactory, Forssman undertook to determine whether some other conditions might be of influence. His experiments were performed as follows: A section of nerve was removed and the two ends then placed in a small tube of straw, sometimes connected by a thread, sometimes allowed to remain separate, and sometimes a piece of nerve resected from another part was placed between them, either within or outside of the straw. He found that in the former case, some of the fibers followed the thread, but many became hopelessly entangled and lost. When, however, a piece of nerve was interposed, even if it lay outside the tube, the nerve fibers grew into it and followed it to the peripheral portion. He then filled the tubes with an emulsion of brain or spinal cord substance, and found that it exerted the same attractive influence, and he therefore concludes that nervous tissue contains some peculiar element that attracts to itself regenerating nerve fibers. This influence he names positive neuro-trophism, and ascribes it not alone to the disintegration of the nerve substance but to the disintegration in a peculiar manner. The degenerated nerve tissue can apparently exert this influence over a considerable space, a condition that he names chemo-trophism.

SAILER.

36. NUCLEUS DIAPHRAGMÆ. ÉTUDE SUR L'ORIGINE RÉELLE DU NERF DIAPHRAGMATIQUE (Origin of the Phrenic Nerve). Fr. Sano (Journal médical de Bruxelles, 1898, No. 42).

In the human subject studied by Nissl's method the author comes to the following results with reference to the phrenic nuclei. The

motor neurons, which enter into the constitution of the phrenic nerve, have their ganglion cells grouped in a long spinal nucleus, which occupies the central portion of the anterior horns, from the lower third cervical segment to the middle of the sixth segment.

The sensory neurons of this same nerve have nuclei of origin in small groups in the spinal ganglia of the third, fourth, fifth and sixth cervical nerves.

The vaso-motor neurons are in relation with the middle and inferior grand sympathetic and at times with the first thoracic ganglion. The situation of their ganglion cells has not yet been located with definiteness.

JELLIFFE.

37. HEMORRHAGE INTO PONS, SECONDARY LESIONS OF LEMNISCUS, POSTERIOR LONGITUDINAL FASCICULI, AND FLOCCULUS CEREBELLI. S. Gee and H. H. Tooth (*Brain*, 21, 1898, p. 1).

The case here reported presents noteworthy anatomical features. The case was one of hemorrhage into the pons in a young woman of twenty-one years of age, associated with interstitial kidney lesions and hypertrophy of the left ventricle. The hemorrhage was on the right side, encroaching slightly to the left. It lay at about the junction of the middle with the lower third of the pons, and bulged into the fourth ventricle. The whole of the right sixth nucleus was destroyed. The hemorrhage cephalad followed, and was limited to the right lemniscus which was almost totally destroyed up to the level of the spinal end of the fourth nucleus. The patient lived nineteen days after the onset of the hemorrhage. The conclusions of the authors are as follows: 1. With reference to the Lemniscus. a. It consists of fibers which ascend only, the lowest origin being the nucleus cuneatus and nucleus gracilis. It increases markedly cephalad, which is due probably to the accession of fibers from the sensory nuclei of the medulla. b. Its destination. As the corpora quadrigemina are approached the lemniscus gives off a dorsal branch, the lateral lemniscus, which appears to end in the corpora quadrigemina. The main or mesial lemniscus proceeds brainwards towards the optic thalamus, and here again appears to divide into two bundles, one of which loses itself in the optic thalamus; the other mass of fibers appears to take a lateral direction dorsally situated to the corpus sub-thalamicum. These fibers may enter the optic thalamus higher up, or they may pursue an uninterrupted course to the cortex. c. Its function is for the transmission of afferent impulses, but it can scarcely be the only path in the medulla and pons for afferent impressions.

2. Posterior Longitudinal Bundles. These consist of fibers running to and from the brain in probably nearly equal proportions. a. Descending fibers which appear to serve as a path of communication between the motor nuclei above and below the lesion. The position of these fibers suggests a further connection between nuclei above and the anterior and lateral horns of the upper cord, including therefore the nucleus of origin of another cranial nerve, the spinal accessory. b. Ascending fibers enter into close relations with the cells of other motor nuclei (fourth and third). There is a free decussation across the middle line at the level of the third nucleus. Though most of the posterior longitudinal fibers seem to be absorbed in the third, yet a considerable number can be seen above it, and these probably enter into the optic thalamus.

3. Flocculus of the Cerebellum. Its white matter consists largely of fibers derived from some higher level, probably the transverse fibers of the formatio reticularis.

JELLIFFE.

PATHOLOGY.

38. EIN FALL VON LEPRO ANAESTHETICA MIT SECTIONSBEFUND (A Case of Leprosy Anaesthetica with Necropsy). Samgin (Deutsche med. Wochenschrift, 30, 1898, p. 475).

Only a few histological investigations of anesthetic leprosy have been made. The disease, in the case described by Samgin, began with chronic rhinitis, and soon pain and anesthesia in the legs and arms were noticed. The body became almost entirely anesthetic. In most anesthetic areas dissociation of sensation existed, and consisted of complete thermoanesthesia and analgesia, with slightly diminished tactile sensation. Toward the end of life the atrophic patches in the skin became confluent. The skin of the fingers was smooth and desquamative, and scars resulting from analgesic panaris were observed. No other mutilations were found. The facial nerve was paralyzed on both sides in the upper portion, and on the left side in the lower portion also. The peroneal nerves were paralyzed. The claw-hand was present. Leprosy bacilli could not be found during life. The diagnosis between leprosy and syringomyelia was made from the thickness and hardness of the ulnar nerves, the paralysis of the facial nerves without other bulbar symptoms, and the irregularly located atrophic areas of the skin, which corresponded to the anesthetic regions.

Marked alteration of the skin was found after death, and leprosy bacilli were present in the skin and nerves, but not in the spinal cord, posterior ganglia or cerebral cortex. Interstitial neuritis existed in the ulnar and peroneal nerves. The myelin had almost entirely disappeared. The examination of the spinal cord in this case was especially valuable, as a similar examination has rarely been made. Samgin found secondary degeneration of the posterior roots and of the columns of Goll. Nerve fibers in the posterior ganglia were also degenerated. The cells of the gray matter of the cord were unaltered. Samgin believes the process began in the peripheral ends of the nerves of the skin.

SPILLER.

39. SULLE ALTERAZIONI CADAVERICHE DELLE CELLULE NERVOSE RILEVABILI COL METODO DI NISSL (On Post-mortem Changes in Nerve Cells as Shown by the Nissl Method). G. Neppi (Rivista di Patologia nervosa e mentale, 2, 1897).

In dogs the author investigated the action of post-mortem changes in the ganglion cells of the cord and of the cortex. They were studied hourly from 6-24 hours after death, and changes did not seem to appear until after the first twenty-four hours, if the tissue was kept at a temperature of about 60° F. The nucleus stained less intensely after twenty-four hours. After forty-eight hours diffuse changes seemed to take place, the nucleus stained less distinctly, though the cell contours were fairly well preserved. At the end of seventy-two hours distinct changes were apparent, the cell contours were altered, more rounded; the nucleus seemed atrophied. Complete degeneration with poorly staining cytoplasm and nucleoplasm took place in ninety-six hours.

JELLIFFE.

CLINICAL NEUROLOGY.

40. CONSERVATION DES RÉFLEXES PATELLAIRES DANS LE TABES DORSALIS (Preservation of the Patellar Reflexes in Tabes Dorsalis). C. Achard and Léopold-Lévi (Nouvelle Iconographie de la Salpêtrière, 2, 1898, p. 83).

Six cases of tabes, without necropsy, and one with necropsy, in which the knee jerks were preserved, are reported. The histological

examination in the last case showed that the preservation of the knee jerks was due to the preservation of the root entrance zones of Westphal in the posterior columns of the lumbar region. SPILLER.

41. ATROPHIE MUSCULAIRE TABÉTIQUE À LA PÉRIODE PRÉATAXIQUE (Tabetic Muscular Atrophy in the Preataxic Period). M. Dejerine (La Médecine moderne, 9, 1898, p. 310).

Muscular atrophy in ataxic patients is encountered at all periods of the disorder, but Dejerine reports from the Salpêtrière a case in the preataxic period. A woman of 44 came to the hospital for clubfoot with atrophy of the common extensor and other related troubles. Two months before, during the night, she was seized with sciatic pain of great intensity. She perceived the next day that there was a paralysis of the muscles supplied by the left external popliteal branch. Upon examination, the sensibility and electrical contractility were found intact. No knee jerk, no Argyll-Robertson pupil. Romberg's sign not to be found, owing to the fact that the woman could not stand upon the right foot. From all these facts and the further history of the patient's having had an attack of lightning pain six years before, and a slight occasional twitch of pain in the lower limbs, the diagnosis of tabes was made. Dejerine does not think the paralysis of tabes incurable, although there is one form which is progressive and entirely rebellious to treatment, the prognosis no doubt being like that of the ocular paralysis of the same disorder, dependent upon whether the lesion bringing it about is peripheral or central. He considers electricity the only treatment worth mentioning. MITCHELL.

42. UEBER EINE BEWEGUNGSPROBE UND BEWEGUNGSSTÖRUNG BEI LUMBALSCHMERZ UND BEI ISCHIAS (Concerning a Movement Test and Disturbance of Motion in Lumbar Pain and Sciatica). By L. Minor (Deutsche med. Wochenschrift, 23 and 24, 1898, pp. 363 and 382).

Minor has noticed that persons not paralyzed, but suffering from traumatic pain in the back, rise from a sitting position on the floor in the same manner that persons do who are afflicted with the pseudohypertrophic form of muscular dystrophy, i. e., they turn their faces toward the ground, extend their arms, place themselves on all fours, and climb upon themselves by supporting the hands upon the lower limbs. Strange to say, this fact has received almost no attention in the literature. This peculiar method of rising is only present when the pain is bilateral, and has been seen in lumbago, and when the muscles of the back have been overstrained. In some cases the pain seems to be due to kidney trouble, or vertebral caries. The cause of this method of rising is the attempt to spare the erector muscles of the trunk.

Minor speaks of the frequent occurrence of lumbago and sciatica together, and of the frequent passing of the former into the latter.

In sciatica also the method of rising from the sitting posture is peculiar, and is exactly opposite to that seen when lumbar pain is present, though the object to be attained—the relief of the extensor muscles of the trunk from contraction—is the same in the two conditions. When a patient with sciatica is placed on the ground and told to rise, he extends fully the sound limb, while the painful one is slightly flexed at the knee. He raises himself with his hands behind his back on the floor, flexes the knees, shoves the pelvis backward, and raises the trunk from the ground by leaning on one hand. When the lumbago is changing to sciatica, the mode of rising varies according to the preponderance of the pain in the limb or back. In rare cases this

method of rising is not always present, and usually in these cases the pain is not severe, or is below the popliteal space; otherwise the method is absolutely characteristic of sciatica. SPILLER.

43. COMPLETE ATROPHY OF THE DELTOID WITH VICARIOUS RESTORATION OF FUNCTION. Kennedy (British Medical Journal, June 11, 1898. 1, 1898, p. 1513).

Complete atrophy of the right deltoid following a subcoracoid dislocation from a fall. Two years after the accident the muscle was still useless, but function at the shoulder was almost completely normal, due to the efficient vicarious action of the trapezius, the supraspinatus, the muscles rotating the scapula and to torsion and curvature of the vertebral column. PATRICK.

44. UEBER EINEN FALL VON HYSTERIE MIT UNGEWÖHNLICHEN SYMPTOMEN (DIATHESE DE CONTRACTURE) UND DEREN BEEINFLUSSUNG DURCH HYPNOTISCHE SUGGESTION (Concerning a Case of Hysteria with Unusual Symptoms, etc). A. Lilienfeld (Deutsche med. Wochenschrift, 27, 1898, p. 426).

A woman of forty-seven years began in 1894 to present, among other signs of hysteria, frequently repeated painful contractions of the abdominal muscles; later the condition developed which is known among the French as "diathèse ou état d'opportunité de contracture." She was unable to walk even a few steps, inasmuch as the slightest irritation, such as striking the foot against a stone or the corner of the bed, or an unexpected noise, caused spasm in both legs, with the production of the equinovarus position of the feet. The calf muscles were especially liable to be thrown into spasm. At times the shoulder muscles were involved, and the arm could not be raised or abducted, or the muscles of the fingers were affected. The contraction of the abdominal muscles alone produced pain. The spasms were easily overcome, but if no attempt was made to control them they persisted. A vigorous passive movement of the part in the direction opposite to that of the contraction, or vigorous use of the faradic current, was usually sufficient. Hypnotism was most effective, and rendered the patient much less susceptible to spasm, but did not produce permanent results. The handwriting, which was almost illegible, became temporarily very distinct by the employment of hypnotism. The patient had great difficulty in speaking, on account of spasm of the tongue, and at times was unable to utter a word. SPILLER.

45. PARALYSIE RADICULAIRE INFÉRIEURE DU PLEXUS BRACHIAL; AUTOPSIE (Paralysis of the Inferior Branch of the Brachial Plexus; Autopsy). M. Apert. (La Médecine moderne, 9, 1898, p. 471).

M. Apert presented to the Société Médicale des Hôpitaux the specimens from the autopsy of a man who had complete loss of power in the right arm for thirty-three years from an injury received by being run over by a wagon. He had previously been presented to the society to exhibit the remarkable atrophy which extended throughout the whole of the arm, forearm, and hand, except the long supinator, the deltoid, and the biceps. The triceps, the pectoralis major, the dorsalis major, were all atrophied. The patient had also the ocular difficulties described by Madame Dejerine, myosis, narrowing of the palpebral opening, and diminution of the pupillary reflex. The patient died of pulmonary tuberculosis, and upon examination the muscles of the arm, except those mentioned, were found completely degenerated, and trans-

formed into fibrous tissue without a trace of muscular element. The nerve trunks at the first glance seemed healthy enough, but, upon section, the degeneration was observed to be complete, and they were almost like empty tubes. In the spine below the first dorsal vertebra no change was observed, but at the level of the first dorsal and of the eighth cervical, there was a diminution in the size of the right anterior cornu, and a great decrease in the number of cells in this column. At the level of the seventh cervical, there was complete disappearance of the stellate cells of the anterior horn. Above this, the structure again became healthy; there was no sign of sclerosis or of inflammatory processes. The white substance displayed no change nor the bulb and protuberance. In the left hemisphere of the cerebrum the ascending frontal convolution was considerably lessened in size, being only about half as deep as that of the opposite side. No sign of disease could be found, the condition being simply one of atrophy. The atrophy of these centers cannot be considered as due to an ascending degeneration at all, but simply as an atrophy from loss of function of an organ, favored by the age at which the injury to the arm took place.

MITCHELL.

46. CEREbellar LOCALIZATION. J. S. Risien Russell (Clinical Journal, 13, 1898, p. 5.)

Within the last six or eight years no one has done more to advance our knowledge of the cerebellum than has the above author, and many of the practical results of his investigations are epitomized in this lecture.

Clinically, cerebellar localization is one of the most perplexing problems that confront the diagnostician. Besides the difficulties inseparable from incomplete knowledge of this part of the brain, there is an added element of uncertainty in the fact that in cerebellar lesions in man we have, as a rule, to deal not only with defects consequent on destruction of cerebellar tissue, and which are strictly comparable to the effects which follow ablation of parts of the organ experimentally, but also with others the result of increase of intra-cranial pressure. The effect of increase of intra-cranial pressure is to distort the clinical picture by adding to it extraneous phenomena in no way dependent on the destruction of cerebellar tissue. This increase of pressure, however, oftentimes responsible for so much confusion, is not entirely an unmixed evil, but under certain circumstances some of its effects are important aids to diagnosis. The present lecture concerns only the signs which indicate the existence of a lesion of one lateral lobe, exclusive of involvement of the middle lobe.

Inco-ordination is the most striking feature noted after experimental ablation of one-half of the organ, and manifests itself in various ways.

Rotation:—At first all other modes of its manifestation may be subservient to rotation, the direction of which is best described in terms relating to a screw. In a right-sided lesion the rotation of a subject is in the direction of a right-handed male screw entering an object, while in a left-sided lesion the direction of rotation corresponds to that of a similar screw coming out of an object. This phenomenon does not always occur after experimental lesions, and is only exceptionally met with in man, a fact that is not surprising in that it is much less pronounced in the monkey than in the dog.

Titubation is always obvious when the tendency to rotation is sufficiently in abeyance to make sitting up or standing possible. The unsteadiness of the head on the trunk resembles closely that seen in disseminated sclerosis, and is increased by any attempt at voluntary move-

ment, e. g., feeding. This phenomenon may be well marked in clinical cases, but not so much so as inco-ordination or ataxy of the limbs best seen in the disturbance of the gait of man. The subject walks with the lower extremities far apart, on a wide base, moving the limbs forward in an unsteady manner, it may be tending to stamp the heels unduly, and evincing more inco-ordination of the limb on the side of the cerebellar lesion than of that on the opposite side. Oftentimes this difference is only very slight in degree, and will only be appreciated on most careful observation. Similarly, the inco-ordination may sometimes be demonstrated as the patient lies in bed and will be present in the upper as well as in the lower extremities, and more marked on the side of the lesion. Both the unsteadiness which is seen most typically in multiple sclerosis, and the ataxia with loss of sense of position that is best marked in tabes, may be evident on both sides or confined to the side of the lesion. When the defect in the upper extremity is very slight the test should be made with the whole arm, i. e., with the upper arm held by the patient at a right angle to the trunk.

Reeling is another phenomenon which is much more constant as a result of experimental lesions than as one of the effects of cerebellar disease in man, and, moreover, while its direction indicates with great certainty the side of the experimental lesion, few of the signs on which we rely for the localization of the seat of disease clinically are less reliable than is this one. Experimentally the subject reels so as to fall away from the side of the lesion, but clinically we sometimes find the patient reeling to the side of the lesion, while in other instances he reels to the opposite side, as in the case of the experimental lesions. This difference, no doubt, depends in some way on the increased intracranial pressure present in addition to the destructive lesion of the cerebellum in most of the cases met with clinically. In attempting to localize a cerebellar lesion the observer should not be influenced by the side to which the patient lurches or tends to fall, unless this sign is in accord with the other phenomena present on which a diagnosis is being based. Where other signs present point to one side of the cerebellum being affected, while the direction of the reeling indicates the other, it is better to disregard the latter sign, and to be guided by the indications which the others afford.

The attitude is very characteristic. The trunk is curved with the concavity to the side of the cerebellar lesion, and the side of the face is approximated to the shoulder on the same side; but in addition to this, in animals, there is a curious rotation of the neck, so that the side of the face corresponding to the cerebellar lesion is turned upwards, and at the same time the chin is directed to the affected side. There has been this difference, however, in the attitude in the cases in which one has seen this sign in man, as compared to what one has found in animals—the chin appears to be directed to the opposite side instead of to the side of the lesion. Otherwise the attitude is the same as after experimental lesions; there is the same lateral inclination of the head to the side of the lesion, so that the side of the face is approximated to the shoulder, and there is the same lateral curvature of the spinal column with the concavity of the curve to the side of the lesion.

A one-sided lesion of the cerebellum may cause rigidity and convulsions limited to muscles on the same side as the lesion, but these symptoms are not of great practical value because their occurrence is comparatively rare and because a lesion such as tumor which may cause pressure on the pons may, because of such pressure, cause rigidity and spasm on the side opposite to the lesion or on both sides of the body.

The tendon-jerks furnish one of the most striking differences of what

obtains in man as opposed to the experimental results in animals. In the latter the tendon-jerks are increased; and while this is true of both knee-jerks, that on the side of the lesion is the more exaggerated. Though a similar state of things is met with in man in some instances, in others there may be no difference on the two sides, while in others both knee-jerks are abolished.

Immediately after the operation of removal of one lateral half of the cerebellum the knee-jerk on the side of the lesion is much increased, while that on the opposite side is greatly diminished, if not abolished. By the following day the diminished knee-jerk of the opposite side to the lesion becomes exaggerated, and by the second day it may be impossible to say which of the knee-jerks is the greater. As time goes on, however, the knee-jerks become less and less active, that on the side of the lesion always remaining brisker than normal as long as the subjects were kept under observation (three months), while the knee-jerk on the opposite side usually returned to or about normal, so that at this late stage it is usually possible to determine inequality of the knee-jerks, that on the side of the lesion being increased and greater than that on the opposite side.

That increased activity of the knee-jerks may persist for a long time after a cerebellar lesion is recovered from in man was well illustrated by a case in which Mr. Dean successfully evacuated an abscess in the right side of the cerebellum, for both knee-jerks remained exceedingly active two years and a half after the operation; but Mr. Dean was unable to decide which of the two jerks was the more increased.

As has already been said, while exaggeration of the knee-jerks occurs in cerebellar disease of man, it is as common to find them diminished or abolished. This diminution or abolition of the knee-jerks in cases of cerebellar tumor in man must in some way be associated with the increase of intra-cranial pressure present in these cases. This is suggested by the following two considerations: In the first place, in experimental lesions in animals where pressure is out of the question, apart from the temporary depression of the knee-jerk on the side opposite to a unilateral lesion of the cerebellum, the rule is, as we have seen, that the knee-jerks are increased. Secondly, tumors of the cerebral hemispheres attended with great increase of intra-cranial pressure, especially if the pressure be rapidly increased, may show great diminution or absence of knee-jerks, just as in these cerebellar cases that we are now considering. Moreover, where diminution or abolition of the knee-jerks is met with, variations in their exact state of activity from time to time not uncommonly occur.

Motor paresis is constantly met with in slight degree experimentally, but is only to be detected exceptionally in man. It assumes the form of a hemiplegia in which the limbs on the side of the lesion are affected and the face escapes; or perhaps it would be more correct to speak of it as a triplegia, in that experimentally a slight degree of paresis is also present in the posterior limb of the opposite side. Apart from the fact that this motor paresis is only exceptionally met with in man, while it is a constant phenomenon after experimental lesions, another notable difference exists between the quadruped and man, in that it appears to be the posterior extremity on the side of the lesion that suffers most in the former, while it is the superior extremity on the side of the lesion in which the paresis has been most evident in cases of the kind which have come under the author's observation in man.

Ocular defects are constant in experimental work, the most constant being a turning outward and downward of the eye on the side opposite to the lesion. Clinically, this variety of squint is rarely met with, but

the author has seen it in cases both of tumor and of abscess of the cerebellum. In consequence of the increase of intra-cranial pressure, ocular paralyses are frequent in cerebellar tumor and may be very confusing if their true significance be not recognized. The opposite sixth nerve appears more prone to suffer from pressure in these cases than is the corresponding nerve on the side of the cerebellar disease, so that weakness of the external rectus of the opposite eye may be present, and the globe may accordingly turn more or less inward, i. e., to the side of the cerebellar lesion. This phenomenon has been responsible for errors in localization, owing to its being assumed that the sixth nerve on the side of the cerebellar disease is more likely to suffer from pressure than is its fellow on the opposite side, so that weakness of one external rectus has been taken to mean that the tumor (the most common condition present in these cases) is situated on the same side of the cerebellum.

Another ocular defect which is of great importance in the localization of cerebellar lesions is nystagmus, either spontaneous or evoked when the eyes are moved from their resting position. When the nystagmus is associated with ocular displacements consequent on pressure on one or other of the nerves which supply the muscles of the eyeballs, the special features which characterize nystagmus, the direct result of the destruction of one-half of the cerebellum, can, as a rule, no longer be recognized. Where no ocular displacement occurs at all or where the displacement met with is that characteristic of ablation of one-half of the organ, and where it is not necessary to invoke secondary pressure on any of the ocular nerves to account for the condition, the nystagmus has very special characters which it is important to recognize. In the first place the nystagmus is lateral; and in the next place when not present spontaneously it may not be evoked at all, or only in slight degree when the globes are voluntarily directed away from the side of the lesion, whereas it is pronounced when the eyes are voluntarily turned to the side of the lesion, i. e., in the direction opposite to that in which the opposite globe is displaced as a direct result of the unilateral ablation of the cerebellum. If spontaneous nystagmus exists, voluntary turning of the eyes to the side of the lesion intensifies it, and the nystagmus may become coarser, the range of movement of the globes becoming greater. PATRICK.

47. CASUISTISCHE BEITRÄGE ZUR HIRNCHIRURGIE UND HIRNLOCALISATION (Clinical Contributions to Cerebral Surgery and Cerebral Localisation) H. Liepmann (Monatsschrift für Psychiatrie und Neurologie, Vol. 3, 1898, p. 407).

Liepmann reports a case of crural monoplegia and convulsions. Trephining failed to reveal the tumor, supposed to be in the leg center. The convulsions had existed for ten years, but a number of cases have shown that these may precede the other symptoms of tumor by many years. Inasmuch as the leg was already paralyzed, it was determined to cut the fibers from the leg center, in order to prevent the convulsions. A cut was made about three fingers' breadth from the median line, an inch deep, and at an angle of about 45 degrees to the sagittal plane. The convulsions did not return during the first five months following the operation, and later were infrequent, but the entire left side of the body was paralyzed. After a time the paralysis became partial and limited to the arm and leg. The senses of position, movement and touch in the arm were so disturbed that the limb was useless. The upper branch of the facial was also paralyzed for a week. No voluntary movement of one side of the face was possible, yet in laughing and crying paralysis of the facial muscles could not be seen. Immobility

of the eyeballs to the left, right-sided ptosis, incontinence of bladder and rectum, existed about two weeks. Intense pain, evidently of central origin, was felt in the left thigh. SPILLER.

48. EXPERIMENTAL RESEARCHES AND EXPERIENCES CONCERNING INFILTRATION ANESTHESIA. H. Braun (*Archiv für klinische Chirurgie*, Vol. 57, No. 2, September, 1898).

This very exhaustive investigation begins with a consideration of the general principles of local anesthesia, of which the author distinguishes three separate kinds. The anesthesia may be purely mechanical; or it may be partly mechanical and partly due to the paralyzing influence of the material employed, or, finally, it may be a true regional anesthesia, due to the specific action of the agent used upon the terminal nerve filaments.

In the course of these experiments a number of the more recently proposed local anesthetics were investigated in conjunction with Dr. Heinze, including guaiacol, guaiaryl, aneson, orthoform and eucaine "A." The author found most of them more or less irritating, and unsuitable for the infiltration anesthesia, at all events; more especially guaiacol, which was intensely irritating, and insoluble in water. Eucaine "A" was the only one that effected a practically useful regional anesthesia; but the author does not think that it is equal to very dilute cocaine solutions either in its local anesthetic effect or in the absence of irritation.

The author then investigated cocaine, the lower limit of effective action of which he found to be at 0.005 per cent. (1:20,000), and finally proceeded to beta-eucaine, a substance closely related to it. The limits of its effective action is 0.005 per cent.; like cocaine the addition of 0.04 per cent. of the drug masks the pain of the injection, and equal percentage solutions of both drugs have the same freezing point. Even 10 per cent. beta-eucaine solutions cause no pain any more than cocaine; the intensity and duration of the infiltration anesthesia is the same for solutions of both drugs of equal percentages; only the spread of the anesthesia beyond the limits of the directly infiltrated zone was slightly slower with a 1 per cent. beta-eucaine solution. "There can therefore be no doubt," the author says, "that cocaine and beta-eucaine are the only two substances to be considered in the selection of a drug for infiltration anesthesia; they alone paralyze without irritation, and without injury to the tissues; and they alone effect an anesthesia lasting enough for practical purposes, even in extreme dilution."

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PSYCHIATRY.

49. ZUR AETIOLOGIE UND THERAPIE DER PROGRESSIVEN PARALYSE. (Etiology and Therapy of General Paresis). R. Seeligmann (*Deutsche Ztschrift f. Nervenheilkunde*, 13, 1898, p. 233).

From a statistical study of cases in the Constanz Asylum the author found that in 130 cases in males, 95 (73 per cent.) had had syphilis, 10 had had a chancre and 25 had never been infected, though some of these were not above suspicion. With reference to more unusual general symptomatology he notes irregular sleeping in the day with persistent insomnia during the night, and cramp-like pains in the abdomen. In some 80 cases, 57 showed reflex immobile pupils, in 18 the reaction was normal, and in those with immobile pupils the knee tendon reflexes were increased in 30 cases and absent in 14; in those with normal eyes a similar proportion was noted, 10 had increased knee

jerks and in four the reflex was absent. The author believes that energetic mercurial treatment may be of benefit in some cases.

50. SUR UN CAS DE PARALYSIE GÉNÉRALE JUVÉNILE A DÉBUT SPINAL (On a Case of Juvenile General Paresis of Spinal Origin). A. Joffroy (Revue de Psychiatrie, 1898, p. 166).

This communication gives the clinical history of a male 24 years of age who had been affected for three or four years past. Difficulty in speech, Argyll Robertson pupils, tremor of tongue and of fingers were pronounced. Memory was impaired, judgment nil and incoherent and absurd ideas of grandeur were evidence of the diagnosis. Another case in a male of 19 is described, and one in a woman of 23 who had been ill for two years. The author states that the characteristic feature in these early cases is the dementia rather than delusional ideas of grandeur. While not disbelieving in the para-syphilitic theory as accounting for the disease, he is inclined to lay more stress on hereditary alcoholism.

51. WELCHE ÄNDERUNG HAT DAS KLINISCHE BILD DER PROGRESSIVEN PARALYSE DER IRREN IN DEN LETZTEN DECENNIEN ERFAHREN (What Clinical Changes Has General Paresis Undergone in the Last Ten Years)? E. Mendel (Neurologisches Centralblatt, 17, 1898).

This subject has received some attention during the past few years, and the changes noted by the various authors would seem to show that some alteration in the type of the disease was manifest. The present author believes that, as a rule, general paresis with simple advancing dementia is more common now than formerly, and that cases with great impulsiveness and delusions of grandeur are fewer. His statistics show that in and about the year 1880 the typical cases observed were of 180 cases, 55 in number, while in 1898 of 194 cases only 24 were of the classical order; whereas in 1880 only 37 of the demented cases were observed, while in 1898 there were 70 of this kind.

He also finds that remissions are commoner now than formerly, and also that the disease is, if anything, on the increase, in which particular its increase among women is to be noted, the proportion in his cases being 4 to 1. General paresis in husband and wife is also commented upon as well as the occurrence of paresis and tabes. Thus in seven cases both had paresis; in six, paresis in the husband and tabes in the wife; in three, tabes in the husband and paresis in the wife, and in four cases tabes in both were found. The author suggests that a change in the syphilitic virus might account for this variation in the type, and that some sort of a change in the virus has taken place is held by prominent syphilographers.

52. L'ATROPHIE DU NERF OPTIQUE PAR RAPPORT AU TABES ET A LA PARALYSIE GÉNÉRALE (Optic Atrophy in Tabes and in General Paresis). M. Klippel (Revue de Psychiatrie, 1898, p. 102).

Optic atrophy causing blindness is by no means a rare symptom in tabes, but the author states that in this respect there is a great contrast in these diseases in that it almost never occurs in general paresis, or if present, it is an essentially different class of affection. Tabes is always an affection of the peripheral neurons, whereas paresis always affects the central neurons. In the one there is distinct blindness, in the other there is psychical blindness, dementia.

53. LE DIAGNOSTIC DIFFÉRENTIELLE DE LA PARALYSIE GÉNÉRALE ET D'ALCOOLISME PARALYTIQUE (Differential Diagnosis Between General Paresis and Alcoholic Paresis). M. de Montyel (*Revue de Médecine*, 18, 1898, p. 109).

The author draws a somewhat fanciful picture of the differences occurring in these two conditions. He describes the paretic as grandiose and open hearted in his general feeling, full of care and goodness to his family, while the alcoholic paretic is cold and selfish and cares nothing for those that are dependent upon him. The one begins slowly and insidiously, the other commences suddenly. These are the most important differences. That they have little value in actual practice is evident.

54. CONTRIBUTION TO THE STUDY OF THE BLOOD IN GENERAL PARESIS. Smith Ely Jelliffe (*State Hospitals' Bulletin*, 2, 1897, Nos. 3 and 4).

From a study of the blood in some twenty cases of general paresis, the author draws general negative conclusions. From his findings it would appear that the blood forming organs are certainly not impaired to any great extent. The only constant feature was a reduction in the amount of hemoglobin; in one case it was reduced to 52 per cent. The specific gravity ranged from 1047 to 1060. There was no distinct leucocytosis. The lymphocytes, by some authors regarded as young cells, were uniformly diminished, and in general the large mononuclear leucocytes averaged high. The eosinophiles varied widely, and in some cases were entirely absent.

55. GENERAL PARALYSIS OF THE INSANE DURING ADOLESCENCE, WITH NOTES OF THREE CASES. Purves Stewart (*Brain*, 21, 1898, p. 39).

The writer presents the histories of three cases of general paresis in patients of seventeen, fifteen and seventeen and one-half years of age, respectively. In one of them he was enabled to obtain an autopsy, and he here reports his findings. With reference to the vexed question of etiology, the author states that its relations to inherited syphilis have been well established, but that acquired syphilis is rare, though it has been recorded in at least three cases. The mental symptoms in young adolescents take the form of a simple progressive dementia. The children become dull gradually, forgetful and apathetic, sometimes stuporose, but may have occasional bursts of passion. Grandiose ideas are usually absent. Mild delusions and hallucinations may exist, but they rarely exhibit the extravagant types of adults. Growth is usually arrested, the genital organs remain infantile in type. Fits or "congestive attacks" are common, and may be the first symptoms of the disease. These may not be attended with convulsive movements, but occasionally there may be attacks of general "trembling" without loss of consciousness. Optic atrophy is not uncommon; the pupils resemble those found in the adult form. Febrillary tremors of the face, tongue and lips are usually present; the articulation is typically slurring and slovenly, and the handwriting resembles that of the adult paretic. General analgesia has been noted in some cases. Motor symptoms resemble those of the adult type. Intention tremor similar to that seen in multiple sclerosis has been observed. Ataxia, spasticity and contractures are noted. The knee jerks vary as with the disease in the adult; they may be lost, exaggerated, or remain normal. The superficial reflexes remain normal, and the sphincters usu-

ally remain unaffected, though with the progressive dulness these may be involved.

In children the disease has varied in duration from six months to eight and a half years. Of twenty-eight fatal cases, ten lasted over five years, and fifteen lasted four years. The microscopical findings do not differ materially from those found in the adult. An excellent bibliography is appended.

56. LA PARALYSIE GÉNÉRALE PROGRESSIVE DANS LE JEUNE AGE (avant 20 ans). General Paresis in the young (before the age of 20). C. Thiry. *Gaz. hebdomaire de méd. et de chir.* 1898, No. 45 (Also Thèse de Paris, 1898).

A thorough search through the literature since 1877 has enabled the author to bring together the histories of some 67 cases of general paresis occurring in the young under the age of twenty. In the majority of his cases a neuropathic family history is to be found and syphilis plays an important factor, especially in its effects upon the general nutrition of these young cases which would seem to predispose them to degeneration of their nervous tissues. He shows that the clinical pictures resemble closely those found in the adult both with reference to the cerebral and spinal symptoms. The general progress of the disease, the author holds, is so characteristic even in children, that no mistakes need be made in the diagnosis. Remissions he thinks do not occur in the young and the prognosis is always of the gravest. There would seem to be no special therapeutic measures available. Thorough antisiphilitic treatment, which, according to the author, may be of some avail in the adult type of the disease, in the young is unavailing.

57. UN CASO DI PARALISI PROGRESSIVA IN UN BAMBINO E TABO PARALISI NELLA MADRE (General Paresis in the Child and Tabes in the Mother). A. Grannelli (*Rivista quindicinale di psicologia, psichiatria, neuropatologia*, 2, 1898, p 213).

The author here reports the clinical histories of mother and child. The father was alcoholic and infected the mother with what was apparently syphilis some three or four years before the birth of the child. At the age of 44 the mother developed a typical case of tabes and at the age of 7, following a severe attack of scarlatina with nephritis, the child showed signs of beginning dementia. Her disposition soon underwent a change and she developed a general fine tremor. Later she had an epileptiform attack and subsequently developed into a more or less typical case of general paresis. The autopsy which was incomplete seemed to verify the diagnosis.

JELLIFFE.

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Original Articles.

MOTOR PATHS IN THE BRAIN AND CORD OF THE
MONKEY.¹

BY E. LINDON MELLUS, M.D.
JOHNS HOPKINS UNIVERSITY.

These experiments² consisted in the removal of a single minute portion of cortex from one hemisphere (invariably the left). The animals were allowed to live after the operation for periods varying from 10 to 40 days, when they were killed, the brain and cord carefully removed, hardened in Müller's fluid and stained by the Marchi method. In each operation the greatest care was taken to secure perfect asepsis, and in no single case was there the slightest trace of septic infection. In all the experiments here reported the portion of cortex excised was taken from the so-called motor area; some from the face, some from the arm, and others from the leg area. The movement controlled by the portion removed was, in each case, determined by faradic excitation, the weakest possible current being used to obtain an uncomplicated movement. As is well known in electrical stimulation of the motor

¹ Read before the Philadelphia Neurological Society, November 28, 1898. For discussion on this paper, see p. 178.

² The experiments here described were done in the Pathological Laboratory of University College, London, and in the Anatomical Laboratory of Johns Hopkins University, Baltimore.

cortex, the stronger the current, or the longer continued, the more complex is the movement. In most cases the removal of so small a portion of cortex, generally from 2 to 4 mm. square, was followed by no apparent disturbance of function. In a few cases, mostly following removal of the area governing movements of the thumb, the operation was followed by slight temporary disturbance of function, rarely persisting more than a few days.

The amount of degeneration following the removal of so small a portion of the cortex was in some cases surprising, and although the effort was made to have the area removed always about the same size, the amount of degeneration, or rather the number of degenerated fibers, varied largely in the different animals. Another noticeable result was the difference in the size of the fibers depending upon the portion of cortex excised. The largest fibers were found following lesions in the highest portion of the brain near the top of the fissure of Rolando, that is, that portion of the cortex governing movements of the great toe. Fibers coming from those areas, however, were not all large, but were mixed with finer fibers in varying proportions. The average size of the fibers appeared to diminish in proportion to the distance of the lesion downward on the convexity of the brain. From lesions on the upper border of the fissure of Sylvius, near the lower extremity of the fissure of Rolando, the area controlling movements of the lips and mouth, the fibers were much finer; while from areas just anterior to this, excitation of which produces the rhythmical movement of mastication, and from the region still more anteriorly where the center for laryngeal movements is situated, the degenerating fibers were very fine, so fine in fact that it was almost impossible to trace their course by the Marchi method. This gradual decrease in the size of the axis-cylinders from above downward would seem to correspond to a similar decrease in the size of the pyramidal cells, as well as to bear some relation to the length of the axones.

The degeneration of what may be called association fibers following all these lesions was very considerable. They were all *subcortical*, as it was impossible to trace any degeneration within the substance of the cortex. This was probably be-

cause intracortical fibers in the monkey have little or no myelin. These subcortical fibers were both coarse and fine, and in some instances could be followed long distances from the lesion. Either coming from or passing to the cortex, they do not run close beneath it, but some little distance in the depth of the white substance. Possibly many of the finer of these fibers represent collaterals of the pyramidal fibers, but many are of a caliber equal to the largest pyramidal fibers proceeding directly from the lesion, and may rather be looked upon as axones arising in the portion of cortex removed. Others may have been subcortical fibers passing near the lesion which came in the way of the knife. In many cases, especially when the lesion was near the fissure of Rolando, these subcortical association fibers were particularly numerous passing under that fissure, connecting the cortex of the ascending frontal with that of the ascending parietal convolution. In no other instance do these experiments show such intimate anatomical relations between neighboring gyri. This is quite possibly due to the fact that most of the lesions were not far from the fissure of Rolando. The distribution of these fibers would seem to bear some possible relation to what Beevor and Horsley³ termed the "absolute area of representation." Thus, in electrical stimulation of the cortex there is a "focus" for a certain definite movement, flexion of the hallux for example, a limited area, stimulation of which with a very weak current produces that simple uncomplicated movement. This focus is the center of a considerable cortical area, stimulation of which produces the same movement, but complicated with other and more complex movements. This larger area they term the "absolute area of representation." The distribution of subcortical association fibers was not, however, limited to such areas, but some fibers could be traced beyond the motor cortex into the silent areas. This was more particularly the case when the lesion was near the border of the motor area. Lesions of the ascending frontal convolution were very likely to give rise to some degeneration in the frontal gyri. Occasionally evidences of slight degeneration were found in the temporo-sphenoidal lobe and in the

³ Beevor and Horsley, *Phil. Trans. Roy. Soc., of London*, B. 1888.

convolutions of the internal surface, but never in the occipital lobe.

In all cases scattered degenerated fibers crossed in the middle third of the corpus callosum to be distributed to the convolutions of the opposite hemisphere. This distribution, though generally less extensive than on the side of the lesion and much less in amount, was apparently quite symmetrical. In lesions of those areas controlling movements in which one would expect bilateral representation, such as the rhythmical movement of mastication, the distribution of blackened fibers to the convolutions of the two hemispheres seemed to be more nearly equal. The proportion of the blackened fibers crossing in the corpus callosum to the amount of degeneration proceeding from the lesion was not always constant. The callosal fibers were in most cases both coarse and fine. Often the coarser callosal fibers were quite as coarse as any found on the side of the lesion, from which it would appear probable that the callosal fibers are not made up exclusively of collaterals.

Proceeding inward or downward from the lesion, there was in each case a tract of blackened fibers consisting of either both coarse and fine fibers in varying proportions or, as before stated, in some cases of only very fine fibers. In passing through the centrum ovale on the way to the internal capsule these fibers became more or less scattered and were not again found as a distinct and separate bundle, but were always more or less mixed in with the healthy fibers. In the upper levels of the internal capsule their position seemed to depend very much on the site of the lesion. In the upper levels of the internal capsule above the level where the fibers begin to come in from the frontal lobe to form the anterior limb, levels in which the capsule has no distinct genu and all the fibers are transversely cut in a horizontal plane, fibers coming from lesions in the upper portions of the convexity, as leg and upper arm areas, were found in the posterior half, while fibers from lesions lower down on the convexity, as hand or facial areas, were in the anterior half. The former fibers passed downward and slightly forward, while the latter passed downward and rapidly backward until in the lower levels of the internal capsule all the degenerated pyramidal fibers coming from lesions

in this series of experiments, facial as well as lower limb fibers, were found well within the limits of the middle third of the posterior limb. Within this area there was but slight tendency to a grouping of the fibers coming from any one portion of the cortex and that was so inconstant that it gave no hint of a law governing their arrangement. Beevor and Horsley⁴ figure a certain methodical arrangement of the fibers in the monkey in the internal capsule based upon the results of electrical stimulation of the cut ends of the pyramidal fibers after the removal of successive segments of the brain from above downward. They group the fasciculi in the internal capsule from before backward, much in the same order and sequence as the excitable areas of the cortex are arranged from the fissure of Sylvius upward. This agrees closely with the results of electrical stimulation of the capsule obtained by Frank and Pitres⁵ in the dog, except that the latter investigators obtained facial movements from stimulation of the most posterior as well as the most anterior of the excitable fibers of the capsule. Beevor and Horsley found constant overlapping of the excitable areas in the capsule, overlapping correspondingly as extensive as that observed in cortical areas. This is explained by the demonstration in these experiments of scattered degenerated fibers over the whole excitable area of the internal capsule following restricted lesions of widely separated areas of the motor cortex. If this arrangement holds good in the human brain, damage to fibers in the anterior portion of the capsule or about the genu, if in the upper levels would give rise to facial paresis; while small lesions a few mm. lower would be quite as likely to affect the upper or lower limb.

A large proportion of the finer fibers coming from these lesions passed from the posterior limb of the internal capsule into the thalamus, into which many of them could be followed for some distance. That these fibers are not collaterals given off by the pyramidal fibers as they passed through the capsule is shown by the fact that the fine degeneration in the capsule disappeared in proportion to the amount of degeneration pass-

⁴ Beevor and Horsley, *Phil. Trans.*, B. 1890, p. 49.

⁵ Frank and Pitres, *Leçons sur les fonctions motrices du cerveau*, etc., par Frank, Paris, 1887.

ing into the thalamus. While these fine fibers were very much mixed up with the coarser fibers in the capsule, they were much more numerous posteriorly, often encroaching considerably upon the posterior third of the posterior limb.

From here on we will follow the degeneration from these lesions to their destination in groups arranged according to the location of the lesion upon the cortex, taking first those degenerations resulting from a lesion in the hallux center. The degeneration had, as stated, suffered a considerable diminution by the passage of fibers, mostly fine, into the optic thalamus. In the crus the remaining degenerated fibers were situated in the middle third, not in a compact mass, but pretty evenly scattered over the whole area. Here they suffered a still further loss, many fibers passing from the dorsal border of the crus into the substantia nigra, where they could be traced but a short distance before they disappeared. The proportion of fibers passing to the substantia nigra varied in different cases. The movement of these fibers toward the border of the crus adjacent to the substantia nigra suggested an inversion of the apex of the triangle which is generally figured as representing the position of the pyramidal fibers in the middle third of the crus in man. In other cases the area containing degenerated fibers was more like an irregular parallelogram, equally extensive on the border of the crus contiguous to the optic tract as on that bordering the substantia nigra. The remaining fibers passed on through the pons and medulla, scattered over the entire cross section of the left pyramidal tract. There was **nothing** to suggest any diminution in the number of degenerated fibers in the passage through the pons and medulla. At the decussation of the pyramids the tract clearly divided, the majority of the degenerated fibers crossing to the lateral column of the opposite side, while the remainder, the proportion varying in different cases, passed to the lateral column of the same side. Some blackened fibers clearly remained in the left anterior column. They retained this position, bordering the anterior longitudinal fissure, throughout the cervical and dorsal cord, their disappearance in the lumbar region apparently corresponding to that of the degenerated fibers from the lateral columns. None of these fibers were observed to decussate.

Although it has been asserted that there is no direct pyramidal tract in animals, these results show clearly that it does exist in some monkeys, and it is quite possible that it varies as much in individual animals as Flechsig has shown that it does in man. At the time that some of these results were first published in the Proceedings of the Royal Society of London,⁶ Muratoff⁷ had already published similar observations upon the dog. The division of the pyramidal tract at the decussation has since been demonstrated in the cat, dog and monkey by numerous investigators, and that the same condition obtains in man has now been proved by the observations of Dejerine, Hoche and Risien Russell. The varying proportion of pyramidal fibers passing to the lateral column of the same side (homolateral as they are termed by Russell) may depend upon varying proportions of the direct pyramidal tract. Further investigation may show that the number of homolateral fibers bears some direct relation to the size of the direct tract. Such relations, however, would suggest a participation of the direct and homolateral tracts in bilateral representation, and this is not borne out by the observations of Hoche⁸ and Risien Russell⁹ in the ultimate decussation of the fibers of the direct tract. The number and relative proportion of degenerated fibers in the crossed tract of each side remained unchanged throughout the cervical and dorsal cord. In the lumbar enlargement they gradually diminished from above downward. Occasional fibers could be traced for some distance toward the anterior horn cells, though the oblique course taken by these fibers as they left the pyramidal tract made them difficult to follow. In no instance could they be traced to the large cells of the anterior horn. Throughout the lumbar enlargement fine fibers were found leaving the crossed tract, and they could be followed across the middle line into the gray matter of the opposite side. Most of these fibers appeared to cross in the posterior commissure. In this connection it may be remarked that Ramon y Cajal¹⁰ states that fibers

⁶ Proc. Roy. Soc., 1894, Vol. 55, p. 208.

⁷ Muratoff, Archiv. für Anat. und Entwicklungsgeschichte, 1893, p. 97.

⁸ Hoche, Archiv. für Psych. Vol. XXVIII, p. 980.

⁹ Russell, Brain, Summer Number, 1898.

¹⁰ Cajal, L'anatomie fine de la moelle épinière, Berlin, 1895, p. 13.

in the lateral column give off collaterals which cross to the other side of the cord in the posterior commissure.

The degeneration following lesions of the thumb center was always proportionately less than that proceeding from hallux centers and consisted, as a rule, of finer fibers. While a certain proportion of these were tolerably coarse, the proportion of fine fibers was also much greater. In these cases the degeneration in the crus was not so fairly in the middle third. As a rule it was displaced toward the outer third, often encroaching on its limits. The passage of degenerated fibers to the substantia nigra was much more extensive, in some cases complete, so that below the crus no degenerated fibers could be found. In those cases in which degeneration could be traced on through the pons and medulla, the number of these fibers grew constantly less. Here and there single blackened fibers could be found leaving the pyramidal tract, but they soon disappeared and their destination could not be demonstrated. In consideration of the proximity of the facial area to these lesions, and the very considerable overlapping of all motor cortical areas, as demonstrated in electrical stimulation of the cortex, it is altogether probable that these lesions destroyed some cells connected with the facial nuclei. This assumption is strengthened by the fact that in some lesions of the facial area as far downward as the upper border of the fissure of Sylvius, a few degenerated fibers were traced into the cervical cord. Only in one instance was there a division of the degenerated fibers at the decussation of the pyramids following lesion of the thumb center. In that case very few fibers passed to the lateral column of the same side, but there were also in the same case a few degenerated fibers in the direct tract along the border of the anterior longitudinal fissure. The remaining blackened fibers in each crossed tract were very evenly scattered over the area allotted to each. These degenerated fibers and the few remaining in the direct tract in one animal maintained their numbers and relations until they reached the level of the seventh or eighth cervical root where they began to disappear. From this level downward to the level of the third dorsal root the degenerated fibers disappeared steadily and gradually, and at the level of the third

dorsal root there were none left. As was noted in lesions of the hallux center, there was a movement of fine blackened fibers from the crossed pyramidal tract transversely across the cord, simultaneous with and corresponding to the disappearance of the degeneration from that tract. Most of these fibers could be traced nearly to the median line, and some could be distinctly seen to cross in the posterior commissure. Such fibers were only seen in those levels of the cord where degenerated fibers appeared to end, from which it would appear that if pyramidal fibers do give off such collaterals, it is only just before their termination. In these experiments the blackened fibers could be followed some distance in the direction of, but never as far as, the anterior horn cells. This could not be expected, the Marchi being essentially a fat stain and only affecting the fragments of broken-down myelin. The finer the nerve fibril and the more attenuated the medullary sheath, the smaller and more widely separated are the fat droplets. Thus, the chain of blackened fat globules would probably disappear some distance short of the terminal arborification of the nerve fiber.

Following lesions of the facial area the degenerated fibers were found in the crus as in the internal capsule scattered over the same area as those from other lesions, that is, in the middle third of the crus. There was no apparent grouping of these fibers toward the mesial portion of this area. In one case of lesion in the Rolandic operculum close upon the upper border of the fissure of Sylvius, and slightly behind the lower extremity of the fissure of Rolando, there was a steady migration of blackened fibers, a few at a time, from the middle third of the crus to the extreme outer border of the lateral pontine bundle, from whence they passed into the capsule of the corpora quadrigemina. This was observed in one instance only. In one instance a few fibers from a lesion of the very uppermost portion of the facial area were observed to pass from the pyramidal tract into the subthalamic region, and in one other case a few blackened fibers were traced to the substantia nigra.

In many cases degeneration from lesions of the facial area could not be traced as far as the pons, although in each case the characteristic facial movement was obtained by electrical stimulation before the removal of the small portion of cortex. In

other cases the amount of degeneration in the pyramidal tract in the pons and medulla was very considerable. The distribution of degenerated fibers to the large-celled nuclei of the pons and medulla was most noteworthy. These fibers began to leave the pyramid in the upper levels of the pons above the level of the motor nucleus of the fifth, where a few single fibers were seen now and then leaving the most dorsal of the pontine bundles of the left side and plunging into the mesial fillet about midway between its mesial and lateral extremities. These fibers disappeared before reaching the dorsal border of the fillet. From this level downward through the pons a few blackened fibers pursued the same course, but the aggregate number leaving the area of degeneration in the pons was small. In the level of the exit of the auditory nerve many blackened fibers began to leave the left pyramid, always as single fibers, mostly from the mesial portion of its dorsal border. They entered the tegmentum and turned downward for a short distance, when they again turned and ran transversely and obliquely through the tegmentum to the neighborhood of the facial nucleus of each side. In no case could a blackened fiber be shown to run into the nucleus or to come in contact with a motor cell. This, of course, would be impossible unless the fiber retained its medullary sheath to its termination. It was only the very uppermost of these fibers that turned downward again after leaving the pyramid. In all the lower levels they passed from the left pyramid directly toward the facial nucleus of either the right or left side. The majority of these fibers leaving the pyramid near the raphe crossed it and ran to the neighborhood of the facial nucleus of the right side. A smaller number of blackened fibers were soon found leaving the pyramid near its external or lateral border. Most of these fibers could be traced almost to the facial nucleus of the same side, but a small number crossed through the tegmentum to a point in the raphe considerably dorsal to that crossed by the fibers leaving the pyramid more mesially, where they crossed and ran toward the facial nucleus of the opposite side. Of all the blackened fibers leaving the pyramid in these levels the majority apparently went to the nucleus of the opposite side. As those levels were reached in which the inferior olive is well de-

veloped no blackened fibers were found leaving the pyramid except from the mesial portion near the raphe. In one instance blackened fibers from the left pyramid crossed the raphe and ran transversely through the right pyramid and disappeared. The majority of these fibers left the pyramid in the levels of the facial nuclei, but occasional fibers passed from the mesial border of the pyramid as low as the decussation of the fillet. These fibers apparently went to the nucleus ambiguus of each side, somewhat in the same proportion as to the facial nuclei, the majority to the opposite side. As before mentioned, a small number of blackened fibers were found at lower levels, even as far down as the seventh or eighth cervical root. This probably explains the arm, hand and finger movements often obtained by stimulation of the facial area. The movements controlled by the cortex are entirely associated movements, and such movements must obviously be provided for either by independent fibers communicating directly with the spinal centers of the various movements, or by association fibers connecting cortical areas. It is altogether probable that the latter method is amply provided for in man, where the development of association tracts is the most remarkable feature of brain anatomy, but these experiments seem to prove that in the monkey pyramidal cells exist in widely separated portions of the motor cortex that may directly control, to a greater or less extent, the same groups of muscles.

LEGENDS.

FIG. I. Horizontal section through the lower level of the internal capsule, showing location and extent of degeneration in the posterior limb following excision of a portion of the cortex from the hallux center of the left hemisphere.

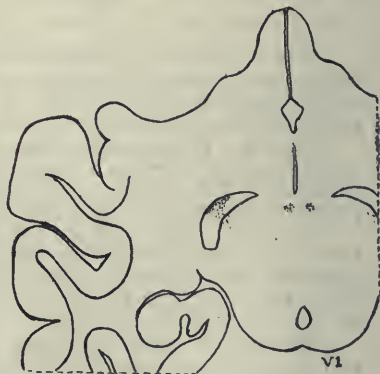
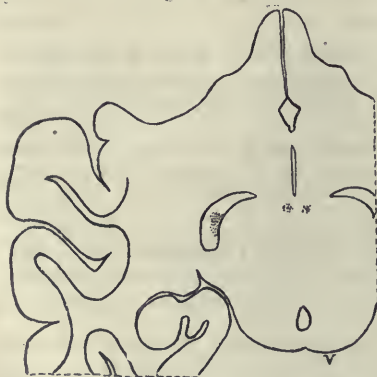
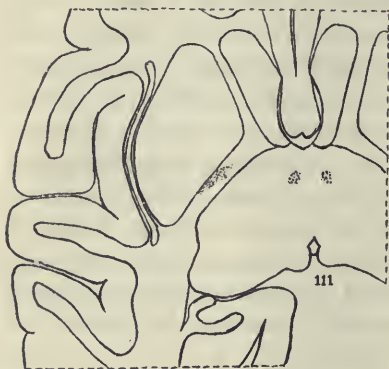
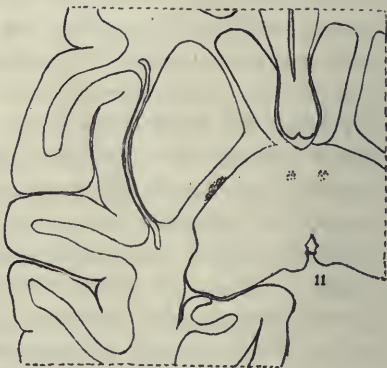
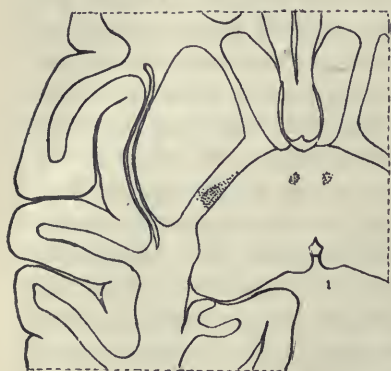
FIG. II. Horizontal section through the lower level of the internal capsule, showing location and extent of degeneration in the posterior limb following excision of a portion of the cortex from the thumb center of left hemisphere.

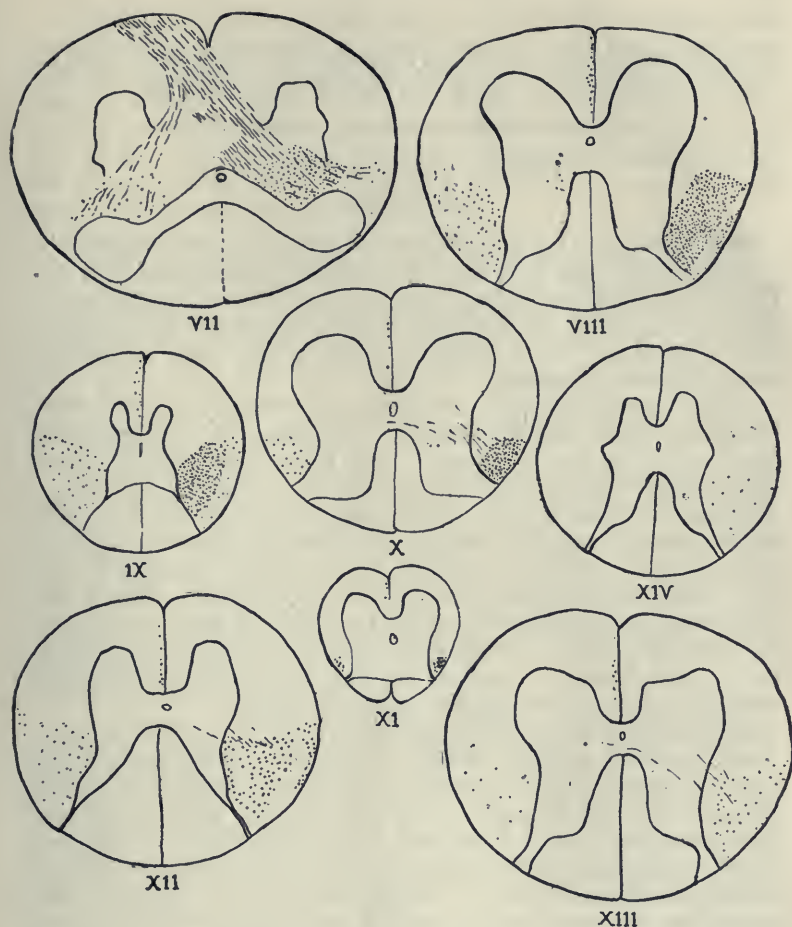
FIG. III. Horizontal section through the lower level of the internal capsule, showing location and extent of degeneration in the posterior limb following excision of a portion of the cortex of the left hemisphere from the upper portion of the facial area just anterior to the Rolandic fissure.

FIG. IV. Horizontal section through the crus, showing location and extent of degeneration following excision of hallux center.

FIG. V. Horizontal section through the crus, showing location and extent of degeneration following excision of the thumb center.

FIG. VI. Horizontal section through the crus, showing location and extent of degeneration following excision of the facial center. Same lesion as in Fig. III.





FIGS. VII, VIII, IX, X, XI. Transverse sections of the spinal cord, showing extent and distribution of degeneration at the level of the respective spinal roots following excision of the hallux center. (VII, motor decussation; VIII, 5 c.; IX, 6 t.; X, 2 l.; XI, 3 s.).

FIGS. XII, XIII, XIV. Transverse sections of the spinal cord, showing extent and distribution of degeneration at the level of the respective spinal roots following excision of the thumb center. (XII, 2 c.; XIII, 6 c.; XIV, 2 t.)

ACUTE ANTERIOR POLIOMYELITIS OCCURRING SIMULTANEOUSLY IN A BROTHER AND SISTER.

WITH REMARKS UPON ITS ETIOLOGY.*

FREDERICK A. PACKARD, M.D.

PHILADELPHIA.

On November 29, Samuel and Hannah G., aged respectively two and a half and one and a half years, were admitted to the Children's Hospital, having been sent there from their home in Royer's Ford, Pa. They had both been exposed to bad weather and seemed to "have caught a cold" at the same time, four weeks before admission.

They are members of an apparently healthy family, there being one other healthy child of this generation, another having died of cholera infantum. The older child, Samuel, had always been well and strong, except for an attack of cholera infantum when an infant. He seemed to be perfectly well until the exposure mentioned above. After this he was a little feverish, and three days later was found to be unable to move his left leg. No other portion of the body was affected. For a few days at the outset of his illness the appetite was poor, but except for this symptom and fever there was no evidence of constitutional disturbance. The loss of power remained unchanged, although his other symptoms rapidly subsided.

On admission he was found to be a large child, well nourished, but with a rather muddy complexion. His head was not typically rachitic or syphilitic, yet it was of decidedly square shape. The pupils were equal and natural, the tongue was protruded straight, and the face was symmetrical. There was slight enlargement of the epiphyses of the wrists and a slightly developed rachitic rosary. Examination of the thoracic and abdominal viscera showed nothing abnormal. The arms were well developed, as was also the right leg. The deep reflexes in these extremities were well preserved and there was no atrophy. The left leg was distinctly cooler than the right and was never moved voluntarily, and the muscles all felt soft and flabby. The knee-jerk on the left side could not be elicited. Measurements of the legs showed the following differences in centimeters:

The right limb at gluteal crease was 26 cm.; the right thigh was $22\frac{1}{2}$ cm.; the right leg was 18.5 cm. The left limb was $26\frac{1}{2}$ cm.; the left thigh was $20\frac{1}{2}$ cm.; the left leg was 18.5 cm.

*Read before the Philadelphia Neurological Society, January 23, 1899.

For discussion on this paper, see p. 240.

On standing him up with support the left leg remained limp and flaccid, and on helping him to walk it was found that the left leg would not support the body, and was swung forward only to a slight degree by the aid of the trunk muscles. The faradic current elicited a faint response in the vastus externus and tensor vagina femoris, but failed to produce contraction in other muscles of the left thigh and leg; whereas on the right side reaction was prompt and vigorous.

Hannah had never been ill until her present trouble began. Following exposure to bad weather for a few hours (at the same time as in the case of Samuel) she was feverish for a few days. Three days after the exposure, a few hours after Samuel's palsy was noticed, she was found to be unable to move her right arm. She had no constitutional disturbance aside from fever and loss of appetite. The arm remained completely palsied from the outset, without involvement of any other portion of the body.

On admission she was found to be well developed and nourished. The skin was slightly muddy in hue, and her head was of the same square shape as was her brother's. Her expression was bright, the pupils were equal and natural, the face was symmetrical, and the tongue was thrust out in the median line. There was a slight rachitic rosary and slight enlargement at the lower epiphyses of the radius and ulna. Thoracic and abdominal examination were negative.

The right arm hung motionless and limp with the fingers in semi-flexion. There was evident diminution in bulk of the deltoid on the right side, and all of the muscles of the affected arm were soft and flabby. No distinct difference in temperature of the arms could be detected. The left arm was moved freely and strongly; the right remained still. The other extremities seemed perfectly normal, and the deep reflexes were readily elicited, but were absent in the left arm. Measurement of the two arms showed no difference except for the forearms, where the left was 0.5 centimeters larger than the right. On the first examination no faradic response was obtained in the triceps or deltoid of the right side, but the biceps feebly responded, and the other muscles (of the forearm) reacted fairly well.

The cases were seen by Dr. Graber, of Royer's Ford, on the day of the development of the palsy, and he writes me that no similar cases have occurred in the neighborhood. He had employed electricity on Hannah's arm and states that there was at first no response, but that later some motion developed.

These cases are reported as an addition to our still incomplete knowledge of the etiology of acute anterior poliomyelitis. The probably infectious origin of the disease is mentioned by

quite a large number of authors, but definite evidence of this theory is still sufficiently scanty to induce me to add the report of these cases, and to make some statements regarding the occurrence of similar proof of infection or remarkable coincidence as observed by others.

In the two cases reported we have three possible explanations for the simultaneous occurrence, viz.: accidental coincidence, the action of toxins resulting from absorption from the digestive tract, and infection from without. The first of these postulates can hardly be accepted; although, of course, it cannot be denied. The second suggestion can only be excluded by the absence of any evidence of common dietary indiscretion, and of other phenomena of intoxication. The third explanation is borne out by the accompanying general symptoms, by the length of time, three days, elapsing between the onset of symptoms and the development of palsy, and by the occurrence of epidemics under the observation of others.

The nature of the infecting agent and its port of entry cannot be even suggested in these cases, especial inquiry on this point having failed to demonstrate the existence of any catarrhal condition, wound or other surface trouble. Dr. Graber saw the cases only after the development of palsy and the subsidence of acute general symptoms. He writes me, in answer to my question, that there was no angina at the time of his visit and no history of its previous presence. It is possible, however, that a slight tonsillitis may have been present and unnoted at the onset.

The occurrence of "family cases" and the simultaneous occurrence of the disease in many neighboring households is discussed by some authors, but by others it is either passed over or merely mentioned.

Gowers¹ states that the influence of heredity in the etiology of the disease is slight, but says nothing as to endemic or epidemic occurrence. Leyden and Goldscheider² quote the reports of epidemics by Medin, Leegard and Cordier, and state that Strümpell in Germany (reference not given) saw three cases

¹ Gowers, *Diseases of the Nervous System*.

² Leyden and Goldscheider, *Nothnagel's specielle Pathologie und Therapie*, 1897.

occurring within one month in the same village, while in a boy living in a neighboring village encephalitis with splenic enlargement followed an attack of measles during the same interval. In two of Strümpell's cases the patients were sisters.

Archibald Church³ says nothing as to heredity or endemic occurrence, while Sachs⁴ simply records the occurrence of the epidemics reported by Medin, Briegleb, Colmer and Caverly.

Holt says that it is rare to find several cases in the same family or to trace any relation to nervous antecedents, but says nothing as to endemics.

Osler⁵ states that epidemics have been described and refers to Medin's report.

Haushalter⁶ quotes Cordier's, Leegard's and Medin's reports, and also mentions one recorded by Pierracini. Aside from this he does not add anything regarding its occurrence in families or neighbors.

Sinkler⁷ mentions Cordier's epidemic, but does not make further comment.

I have read all of the reports of epidemics or coincident family occurrences, that were at my command; others I have quoted from abstract, translations or, where neither original article nor abstract was available (as in Briegleb's, Meyer's, Simon's, Pierracini's and Bergenholtz's reports), I have taken as exact the figures given by the authors that I have found who quoted them. In the case of some of these quoted cases the reference given was erroneous. This was the case in that of Meyer (quoted by Erb), of Simon (quoted by Mary Putnam Jacobi).

Hammond⁸ records the simultaneous occurrence of the disease in two brothers after they had been lying on the damp ground.

Meyer (as quoted by Erb⁹) records its occurrence in twin brothers after measles.

³ Church, Archibald, *The American Text-Book of Diseases of Children*.

⁴ Sachs, *Nervous Diseases of Children*.

⁵ Osler, *Practice of Medicine*, p. 942.

⁶ Haushalter, *Traité des mal. de l'enf*, Vol. IV, p. 701.

⁷ Sinkler, *Keating's Cyclopedia of Diseases of Children*, Vol. IV.

⁸ Hammond, *Diseases of the Nervous System*, 6th ed., 1876, p. 451.

⁹ Erb, *Ziemssen's Cyclopedia*, Vol. XIII, p. 669.

Simon (quoted by Mary Putnam-Jacobi¹⁰) reported three cases occurring in one family practically simultaneously, two being attacked one day and the third twenty-four hours later.

The epidemic of nervous disease reported in a brief note by Colmer¹¹ was possibly due to poliomyelitis, although the note does not definitely establish its nature. It occurred in a parish in Louisiana. It is described in almost these words: A child was seen who was slowly recovering from an attack of hemiplegia. The *parents* stated that within the preceding three or four months eight or ten other cases had occurred within a few miles of their residence. All of the cases had recovered completely, or were improving, all were younger than two years of age, and all were teething. It is, of course, questionable whether this was not an epidemic of cerebro-spinal meningitis. I mention it here as it has been quoted by various authors.

Cordier¹² reports an epidemic occurring at Sainte-Foy in which thirteen cases developed in a village of 1,400 to 1,500 inhabitants during the months of June and July, 1885. Two occurred in brother and sister. The ages of the patients were from one to thirty months; no older children or adults being affected. The palsy appeared on the second or third day of the illness in many of the cases. Of the thirteen patients four died.

Medin¹³ in 1890 made a full report of a Swedish epidemic of palsy in children that is most striking. Ordinarily he saw but one or two cases in a year; but in 1887 two cases were seen in May, one in June, two in July and then thirty cases between August 1 and September 23. Nine more cases were seen before the end of November. In other words, 44 cases of paralysis in children were seen in six months of that year. In but one family did two cases occur. His table of these cases (somewhat abridged) shows the following:

Poliomyelitis ant. acuta, with paresis of extremities...	27
Facial monoplegia.....	3
Acute polyneuritis	6
Poliomyelitis, with paresis of various cranial nerves...	7
Polioencephalitis, with paresis of abducens.....	1

¹⁰ Pepper's System of Medicine, Vol. V, p. 1151.

¹¹ Colmer, Am. Jour. Med. Sc., 1843, n. s. Vol. V, p. 248.

¹² Cordier, Lyon médical, Jan. 1, 1888.

¹³ Medin, Verhandlungen des x. internationalen medicinischen Congresses, Aug. 7, 1890.

Of these 44 cases poliomyelitis was present in 37 patients.

In 1896 Medin¹⁴ published a second paper recording further observations. Between the years 1888 and 1894 he observed 29 sporadic cases, an annual average of between 3 and 4. In 1895 he saw 21 cases of infantile palsy, 13 of which were typically due to acute anterior poliomyelitis. In both of these epidemics nuclei of cranial nerves were affected in a remarkably large number of cases.

Chr. Leegard¹⁵ (as given in the abstract, the original being unobtainable and untranslatable by me) quotes cases observed by Oxholm, and reported in a Norwegian journal in 1896. These occurred in the persons of five healthy children living in one neighborhood, between the end of July and early in September. No more than a single case occurred in any one house. Later four other cases occurred in the same neighborhood. According to the abstract, Leegard related many examples of the almost simultaneous incidence of the disease in two people in the same household.

Caverly¹⁶ reported an extensive epidemic occurring in 1894 in Otter Creek Valley, Vermont. The same epidemic is also the text of Macphail's paper.¹⁷ In Otter Creek Valley (which comprises an area measuring fifteen by thirty miles) there were thirteen towns with an aggregate population of 26,000. Among these towns 126 cases occurred within a brief term. In some cases an urticarial or erythematous eruption was present; in some (as in Medin's epidemics) cerebral nerves or nuclei participated. In some there was hyperesthesia. In only one instance was there more than one case in a family. During the time of prevalence of the epidemic many domestic animals (horses, dogs and fowls) also died paralyzed. Of the human beings 18 died.

Last year, one of our members, J. Madison Taylor,¹⁸ reported an epidemic occurring in a village of 1,500 inhabitants.

¹⁴ Medin, Nord med. Ark., 1896, N. F. VI, 1, No. 1, Abstracted in Neurologisches Centralblatt, 1896, Vol. XV, p. 1119.

¹⁵ Leegard, Sep. aftryk af "forh. paa det norske lægemøde i Bergen," Abstract in Neurologisches Centralblatt, 1890, Vol. IX, p. 760.

¹⁶ Caverly, New York Medical Record, Dec. 1, 1894, p. 672.

¹⁷ Macphail, British Medical Journal, Dec. 1, 1894, p. 1233.

¹⁸ Taylor, Philadelphia Medical Journal, Jan. 29, 1898, p. 208.

Four cases (two being brothers) were seen, and three more were heard of, while in a neighboring town two other cases had occurred.

Fr. Schultze¹⁹ has recently reported a case of great interest in connection with the subject of the etiology of acute poliomyelitis. The illness began with sudden palsy involving both arms. Thirteen days after the onset lumbar puncture was performed and fluid was obtained containing the Weichselbaum-Jäger meningococcus, without leucocytes. In this case the illness appeared shortly after trauma.

From the simultaneous occurrence of cases in two members of the same family, even though this be somewhat rare, and especially from the epidemic occurrence of the disease, it must be conceded that acute anterior poliomyelitis may be due in many instances, at least, to infection. Whether this infection is of a special or general kind cannot be determined, but that the action of toxins could produce the anatomical changes seen in this disease is not at all unlikely in view of our knowledge of their destructive power in other parts as well as in the central nervous system.

¹⁹ Schultze, Münch. med. Wochensch., Sept. 20, 1898.

References to articles upon the subject that were unobtainable by me at the time of writing were as follows: Briegleb's Inaugural Dissertation (Jena, 1890, referred to by B. Sachs, Nervous Diseases of Children, p. 303); and Pierracini's (*Lo sperimentale*, September, 1895), and Bucelli's (*Policlin. Rev.*, 1877) articles, references to which are appended to Taylor's article, and to the former of which Haushalter refers.

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58. LES TROUBLES TROPHIQUES DE LA PARALYSIE GÉNÉRALE. P. Cololian (*Archives de Neurologie*, 5, 1898, pp. 21 and 177).

Whereas the trophic disturbances of general paresis have often been described, there has been little systematic work done upon them and much variation in the observations. The present series of observations were made upon 57 cases, 33 men and 24 women, all of whom had trophic affections. The most common affection was alopecia, which was present in 61 per cent. of the cases; the next frequent trouble was with the teeth, which were affected in 19 cases, the nails in 17 being discolored or atrophied, or enlarged, or lost. Ichthyosis was present in 11, and sacral scars in 8. Bullous and pemphigus eruptions were noted in 5 cases, zona in 3, and perforating ulcer in 2.

A CASE OF HYSTERIA IN WHICH A NEEDLE-PUNCTURE WAS FOLLOWED BY TYPICAL SYMPTOMS OF ASCENDING NEURITIS.¹

BY J. TORRANCE RUGH, A.B., M.D.

The following case presents points of interest to the neurologist, dermatologist and the general practitioner. To the first, the chief point of interest is the intimate relationship existing between the hysterical and the psychic state; to the second, the peculiar character of the skin lesions; to the third, the marked similarity of the symptoms to those of a traumatic ascending neuritis. The history is as follows:

The patient was a female, aged twenty-two years, a clerk. The family history is good, both parents being alive and well, though the mother has always been of a very nervous disposition. The patient in early life was neurotic, and between the eleventh and fourteenth years of her age had very frequent hysterical spasms, following an injury received by being pushed down a number of steps at school. She suffered much from her spine for several months afterward, but recovered entirely from all pain and ill-effects. There is no doubt as to the nature of the spasms which were very accurately detailed by the patient's mother, who also stated that they usually followed an outburst of temper when she was not permitted to do as she wished. After puberty these ceased, though she infrequently became very much excited, and then she usually developed mild delirium; with these exceptions, her health has been good until the present illness which dates from September 23d, 1898. During the past summer and fall, she had been under a severe strain from a mental and moral shock at the hands of a valued friend, and her health was much impaired thereby.

On September 23d, while using the sewing-machine, the needle pierced the forefinger of the left hand, entering on the radial side, passing obliquely downward under the nail and having its exit on the ulnar side about three-eighths of an inch from the end. When the needle was withdrawn there was no bleeding and no pain. A day or two later she heard reports of cases in which a similar injury resulted seriously, and on the evening of that day pain began in the finger end and

¹ Read before the Philadelphia Neurological Society, December 19, 1898.

gradually increased in severity until in two days it was extreme, and involved the entire arm to the shoulder. The third phalanx of the finger became swollen as did also the radio-dorsal surface of the hand. These parts were also red, hot, tender to the touch, and painful to any motion or pressure. Her physician advised opening the original wound, but as that part was not swollen, she would not consent to it. Poultices and unguents were applied, but to no advantage. Cold water, however, gave her complete relief from both swelling and pain for a week or more. During this time she could not work; she slept poorly, had no appetite, suffered with headache, especially in the occipital region, and was generally depressed.

When I first saw her (October 9th), two and one half weeks after the accident, she had just had a marked chill. Her temperature was 102° , her hands and feet were cold, and she was mildly delirious, but, during lucid intervals, complained bitterly of the pain in the back of her neck and the heaviness of her head. Heat to the head and extremities and a mustard plaster to the back of the neck, with trinitrin, gr. 1-100, by mouth, soon relieved her. She was also given grs. xx of potass. brom. as a hypnotic. On examining the arm the following features were noted:—The proximal phalanx of the index finger was red but not swollen; there was a large, red, edematous spot, about two and one half inches across, on the back of the left hand between the first and fifth metacarpal bones; also two similar spots on the radial and extensor surfaces of the forearm, and another on the outer side of the upper arm. These were uniformly erythematous, edematous, raised above the surrounding skin, had sharply defined edges, were without any appreciable thickening or induration, were hot and painful to the touch and did not pit on pressure. Excepting in the finger, there was no disturbance of sensation or motion and no enlargement of the axillary glands or of any of the lymphatics. A superficial infection, or more probably an ascending neuritis of the peripheral branches of the radial, was diagnosed and a hot solution of lead-water and laudanum was applied constantly for several days until the swelling and soreness had subsided. In a few hours, she became quiet and rested quite well the rest of the night. The next day her condition was much the same, except that her stomach refused to retain anything, even rejecting water as soon as swallowed. In the evening, on examining the arm, I was surprised to find the spots changed from the red color to a mottled blue, looking much as if the fingers or thumb had been used to bruise the parts. There were within the area occupied by the red spots five or six dense blue marks, or more properly speaking, deeply shaded blue marks, for between these dense spots, the

tissues were slightly discolored. She also directed my attention to a sore spot directly under the left breast, which was the same in shape and color as those which had been on the hand and arm. It also possessed the same characteristics as the others, being painful, tender on pressure, red and edematous, but not indurated. In all of these spots, there was at first local elevation of temperature and this same was true of those which appeared subsequently. The same application was used on the spot beneath the left breast as on the former ones. Sulfonal gave excellent results as a hypnotic, and the next day found her much improved in her general condition, but very nervous.

By evening, the spot under the left breast had changed to the same mottled blue color as the others, and another red one appeared under the right breast, running the same course as those on the arm had done; the latter by this time had faded much and a few days later changed to a yellowish color, which persisted for a week or ten days. She remained in bed for a week, using the lead and laudanum solution upon the sore spots and taking the compound syrup of the hypophosphites as a general tonic.

At the end of the week the swelling had disappeared from the arm, pain was only occasional, and soreness was much lessened; her appetite was much improved and she slept all night without any drug. The arm was then rubbed every day and she began to go out of the house.

On October 23d, pain again began in the hand and arm, radiating to the shoulder; and the finger, dorsum of hand and entire arm became red and tender. The three tinctures (tincture of opium, tincture of belladonna, aa f 3 ii ; tincture of iodine f 3 ss) were applied locally three times a day, and the arm was put on a splint. Improvement was very decided under this treatment, and at the end of another week a silicate-of-soda splint was substituted so as to have a lighter and more serviceable dressing. At this time, a large red spot appeared over the left supra-spinatus muscle, and later changed as the others had done. From this time on, the arm grew steadily worse, becoming more swollen and painful. Tincture of iodine was used locally to make more decided counter-irritation, and on November 2d, a cantharides blister, 2x2, was applied just below the elbow on the radial surface of the arm. The arm was so swollen that the silicate splint had to be laid aside and the board used. Her sleep was much disturbed by the pain and soreness of the arm.

On the day following the application of the blister, there was considerable disturbance of sensation in the entire arm and slight loss of power; the entire upper limb was swollen and angry looking. That night grs. xx of sulfonal did not put

her to sleep, but about twelve o'clock all pain ceased and the next day there was complete anesthesia of the arm below a line drawn about the shoulder joint from the apex of the acromion process and apparently complete loss of power. She felt "as though the arm was dropping away from her," and it had every appearance of an infected part, but there was no glandular involvement nor was there any constitutional elevation of temperature beyond a half degree or so. Dr. Wm. G. Spiller then saw her in consultation. He carefully examined her and took the electrical reactions of the limb, and finding no signs of degeneration and undoubted evidences of hysteria, he stated his opinion that the case as it then existed was one of hysteria. A pin was introduced into the arm a quarter of an inch without being felt by the patient, and its withdrawal was not followed by any bleeding, the hole remaining patent for a minute or so. The muscles responded almost normally to both the galvanic and faradic tests, requiring merely a stronger current on account of the swelling of the part. A strong faradic current was then passed through the arm and she was strongly impressed by the suggestion that her condition was not serious, would disappear and would not return. The splint was removed and all medication stopped. To reinforce the suggestion of recovery, a "strong" liniment of pure olive oil was given her to apply once a day without rubbing. Faradism was used every day, and by the fourth day the swelling had entirely disappeared, sensation was completely restored, and muscular power regained. With the exception of slight outbreaks of redness, swelling, or pain, she has remained well, and these outbreaks have been entirely dissipated within twenty-four hours by the use of faradism and suggestion. Another red spot appeared some weeks later over the left breast but soon disappeared. She has resumed her work in the office and is rapidly regaining good health.

The several points of especial interest in this case are: (1) The hemorrhagic eruption; (2) The differentiation of the functional neurosis from a true neuritis, and (3) The possibility of the occurrence of ascending neuritis following a non-suppurative traumatism.

The eruption was somewhat out of the ordinary, though there may be present in hysteria any of the lesions of the skin seen in other diseases from an erythema to a localized gangrene. Van Harlingen² published a very interesting classification of the hysterical neuroses of the skin,

² Van Harlingen, Amer. Jour. Med. Sciences, July, 1897.

and cited some very rare and interesting cases. According to his description, this case was one of localized purpura or hemorrhagic ecchymosis, confined more especially to the distribution of the radial nerve. I have no doubt, as suggested by Dr. Spiller, that severe hemorrhages could be caused in this case by either direct suggestions or by auto-suggestion. The eruption began as a slightly edematous erythema and changed to the purpuric variety, the lesions looking as if they were the result of traumatism. There were at first the signs we usually recognize as those of active inflammation: heat, pain, swelling and redness, but the disappearance was not followed by desquamation of the skin, as has often been observed in true inflammation.

Marinesco³ has called attention to the influence of the brain over the vascular system. He says that such an influence can not now be doubted. Centers must exist in the Rolandic region which control the functions of the vessels, as lesions of this zone are followed by vascular disturbances. Hysteria especially offers evidence of this fact. The muscular atrophy of hysteria, the cutaneous or visceral hemorrhages appearing as the result of subconscious action, demonstrate the influence of the brain upon the vessels. He mentions a case at the Salpêtrière, in which to demonstrate the relation of various hysterical phenomena to auto-suggestion, Charcot placed two hysterical patients side by side, one of whom had blue edema of the lower limb. He ordered the other patient to observe attentively the edematous limb, and after some time this other patient also presented a similar edema. The only explanation of such a phenomenon is that the brain has control over the circulation, and this explanation will also apply to the other forms of disturbances met with in these patients. The higher brain centers are capable of influencing the lower centers sufficiently to produce marked pathological changes in the parts under the control of these lower centers; and when these same higher centers are stimulated by another form of impulse (as by healthy suggestion), they induce in the lower ones regenerative and reparative processes, as rapidly as they previously did the degenerative and diseased ones.

³ Marinesco, *Semaine médicale*, No. 55, p. 465.

The marked similarity of the symptoms in this case to those in a case of ascending neuritis rendered the diagnosis extremely difficult. As mentioned above, disturbances of the mental state, i. e., disturbance of the higher brain centers, may induce functional disorders which closely simulate and are, at times, indistinguishable from organic changes due to other causes, and the differentiation of the conditions may be a matter of great difficulty. In this case there was an injury, followed in three days by heat, swelling, redness, pain and disturbance of function, and the part involved corresponded almost accurately with the cutaneous distribution of the radial nerve. There was tenderness on pressure over the erythematous spots, but not elsewhere, all of which symptoms are characteristic of neuritis; however, the mental emotion to which the patient had been subjected, the existence of the spots under each breast, the anesthesia in sleeve form, the transitory paralysis of the entire limb, the tenderness in the ovarian regions, the history of previous hysterical attacks, and above all, the recovery within three or four days after the use of suggestion, clearly separate the functional from the organic condition.

In all cases of serious results following injuries of various degrees of intensity, the possibility of hysterical complications must be borne in mind, and the numerous coexistent stigmata sought as confirmatory evidence, or eliminated in the cases of organic diseases from other causes. The causes of the organic and functional conditions are much alike, and especially so in that traumatism is frequently present in both; but in hysteria the severity of the symptoms is not in proportion to the injury, but is commensurate to the shock or fright sustained. In my case, the injury was but slight, yet the patient's nervous system was so weakened by her previous experiences that the hearing of fatal results from a similar injury was sufficient to give rise to the subsequent conditions.

One other very important diagnostic point should be mentioned, and that is suggestion. Though a case may be clinically a typical neuritis, yet if it yields to suggestion, either conscious or hypnotic, it is a neurosis pure and simple. The use of this agent is a method of treatment, but in a border-line case it becomes a method of diagnosis as well, and is to be employed

in all those doubtful cases in which a so-called non-septic wound or injury has antedated a short time the onset of the trouble.

There has been much controversy among neurologists as to the possibility of ascending neuritis following a so-called simple non-infected wound, such as this patient had. The earlier writers believed in traumatism as an etiological factor in producing neuritis, and especially those injuries of the nature of punctures and stab. Erb⁴ wrote: "An extension of the disease [neuritis] from its original seat not infrequently takes place, both downward and upward. The more important extension in the centripetal direction occurs either uniformly and continuously or in a sudden metastatic manner. In some cases, again, whilst certain parts of the nerve retain their normal character, others present circumscribed hyperemia, swelling and hypertrophy of connective tissue (Froriep, Rokitansky, Tiesler and others), and in this way the spinal cord may ultimately become affected (Tiesler, Feinberg)," and he recognized traumatism as one of the causes of the original injury. Former writers, however, were not generally particular to mention the part which infection plays in these wounds, but recently quite a discussion has arisen upon this point, and the question has been considered at some length by prominent men on both sides. Strümpell⁵ believes that ascending neuritis only follows open wounds, such as stabs, cuts and gunshots, which are infected, and cites the experiments of Rosenbach and Kast to prove that perfectly aseptic wounds never cause spreading nerve-inflammation, and that subcutaneous injuries, as blows, pressure or luxations cause simply a mechanical disturbance of the nerve-elements, from which follow secondary degeneration, overgrowth of connective tissue and final regeneration. He admits that a neuritis may result from an aseptic wound, but maintains that such a lesion always subsides and shows no tendency to spread to other parts.

J. K. Mitchell (*loc. cit.*) calls attention to the fact that some time always elapses between the receipt of the injury and the

⁴ Erb, Ziemssen's Cyclopedia.

⁵ Strümpell, quoted by J. K. Mitchell, in "Remote Consequences of Injuries of Nerves," 1895.

beginning of the neuritis, and believes that this fact is significant of an infectious origin. This statement, however, is equally true for neurotic cases simulating inflammation, as the above history shows; for in most instances the patient learns of serious results following such injuries after the injury has been received, and several days may elapse before the characteristic symptoms develop. J. K. Mitchell also draws the following conclusions in regard to this condition:

1. Pressure, however brought about, whether by inflammatory exudate, or external injury by blow or weight, may be looked upon as a frequent factor, though not a constant one, in the production of spreading nerve-inflammation; but the presence of inflammation in the surrounding tissues, even in direct contact with the nerves, exerts, curiously, little bad influence.

2. The larger nerve trunks are more prone to present the phenomena of spreading inflammation after injury than small ones.

3. Neuritis may spread either centrifugally or centripetally, the latter in traumatic cases being much the more common form. He believes that the possibility of the ascent and descent of neuritis (though of rare occurrence) must be remembered in the treatment of nerve-traumatisms.

Drs. Howell and Huber conducted a series of experiments upon animals for the purpose of studying the process of repair of nerve-tissue, and in not a single case did subsequent examination show any evidence of neuritis having followed the traumatism occurring in the course of their operations, and this was probably due (as suggested by Mitchell, *loc. cit.*) to the fact that their work was done with strict asepsis.

S. G. Webber,⁶ of Boston, recently reported a series of cases of localized neuritis, and states his belief that a traumatism may excite ascending neuritis, either continuous or metastatic, but in his cases does not note the presence or absence of septic conditions. He mentions three cases of injury to the hand or finger in which ascending neuritis seemed to follow. The first followed a severe burn of the hand by nitric acid, and the neuritis affected the whole arm and shoulder, and was followed by

⁶ Webber, *Bost. Med. and Surg. Jour.*, Vol. CXXXIX, No. 18.

loss of power in the arm (whether complete or not is not stated). The second was one in which the symptoms followed unaccustomed pressure on the radial border of the forefinger during the process of raising a window. In this case, however, he states that a typical hysterical condition developed, which, of course, throws doubt upon the presence of ascending neuritis. The third case "had trouble in the whole arm from hitting the end of the middle finger;" the history is almost too incomplete to judge as to the true nature of the trouble, but as the patient was a woman and the injury slight and without any wound, the condition may have been hysterical. Of the three cases reported, but one seems to be a true neuritis.

A very complete record of a case of ascending neuritis of infectious origin was recently published by Marinesco.⁷ The patient suffered from gangrene of the leg, the result of some irrelevant cause, and sections of tissue taken from the gangrenous area showed the presence of streptococci with leucocytic infiltration and inflammatory degeneration of the nerve-fibers. Sections made from these nerves, taken from a region higher up the leg, also showed leucocytic or degenerative infiltration, but no streptococci. Sections of the cord were also made and degeneration of nerve-cells with leucocytic infiltration of nerve-fibers was found, but only in the region and side of the cord from which those nerves had origin. This seemed to show that the inflammation either had spread primarily and continuously along the nerves of the part, or was secondarily induced by the action of the toxins generated in the diseased area, with the evidence much more strongly in favor of primary spreading. This paper was read for Marinesco by Leyden, of Berlin, who said that he considered this case was positive evidence of the existence of ascending neuritis, following peripheral nerve disturbance, a fact which has long been contested. He also spoke of other cases of ascending neuritis of purulent origin which have recently been reported, as confirmation of the above observation.

In the discussion which followed the reading of this paper, Oppenheim⁸ said he believed the condition a very rare one; he

⁷ Marinesco, *La Presse médicale*, No. 96, November 23, 1898.

⁸ Published in the *Vereins-Beilage*, No. 14, der *Deutsche med. Wochenschrift*, May 12, 1898.

had never seen a case of ascending neuritis involving the spinal cord. He further said he had seen but few cases of ascending neuritis, and even these were uncertain. He believed that most of these cases of so-called ascending neuritis were purely functional, being neuralgic, rheumatic, hysterical or neurotic. He mentioned one case of apparently undoubted neuritis ascendens following the running of a rusty needle into the finger with subsequent pain along the course of the ulnar nerve, but the symptoms yielded to suggestion. He knew cases of ascending neuritis had recently been reported following suppuration, showing that the condition might result from a focus of infection from suppuration, and in these the cord might possibly be secondarily involved, but he was skeptical in regard to such involvement.

Remak, in the same discussion, said he was not so skeptical as Oppenheim in regard to the spreading nerve-inflammation following an infective traumatism, but he was dubious as to its following a traumatism without the presence of suppuration.

Toxic substances circulating in the system have long been recognized as a cause of nerve-inflammation, and it is generally admitted that toxins from a focus of infection and suppuration may act in the same way; when, however, the affected nerve or nerves lie in close relation to the focus of infection or suppuration, the assignment of a cause (toxic or simply traumatic) may be a very difficult matter, though the natural inference would be that the toxins had acted directly upon the nerve structures, an inference which is also supported by the anatomical arrangement of the lymphatics and nerve sheaths. In every injury which causes a solution of the continuity of the tissues, there is local death of the edges of the wound, and while toxins are necessarily present in such a part, it is highly improbable that they act as a cause of neuritis ascendens, unless pus is actually present. Such an inflammation from such a cause has never been observed, though its possibility must be admitted.

CLINICAL NOTES ON A CASE OF SYRINGOMYELIA.

FROM THE CLINIC OF PROF. M. ALLEN STARR, COLUMBIA UNIVERSITY.

REPORTED BY SMITH ELY JELLIFFE, M.D., CLINICAL ASSISTANT.

The patient, C. O'B., now aged 22, first entered the Vanderbilt Clinic in August, 1896. His case at the time presented the clinical picture of an atrophy of the Aran-Duchenne type. He was then lost sight of, and did not return to the clinic until January 16, 1898, after a lapse of two and a half years, when he presented a most interesting condition.

The family history is mainly negative. The father, who was a painter, died at the age of 52 of some tubercular trouble. His son never knew of his having any lead poisoning. The mother is still alive and in good health. One brother died of phthisis and alcoholism, and one sister died in childhood of causes now unknown. In neither mother's nor father's side of the family could any hereditary factor be found.

The patient had always been a healthy boy; he had had some of the usual children's diseases, measles and whooping-cough, but had lost but little time from school up to his sixteenth year, when he went to work in a printing office. Here his work was largely mechanical, and of late years he has been bearing heavy weights (forms) on his left shoulder, carrying them frequently up three flights of stairs. About the age of 18 or 19, some time after the first symptoms of the disease were noted, he had an attack of what he designated as "fever and ague," but no positive history could be elicited. He is not a drinker, denies syphilis, and has led a comparatively regular life.

His present trouble began three or four years ago, though he is unable to localize the onset with exactness. One of the earliest symptoms noted was a beginning stiffness of the fingers of the left hand. This prevented him from closing the hand as readily as formerly. At the same time he felt some paresthesia of the ulnar side of the left hand and arm, and the muscles of the hand commenced to "dwindle away." Some dull pain was felt in the hand and arm. In addition to this he found that it was harder to keep his fingers warm. Sores formed very readily upon his fingers and healed only after three or four months. He attributed a beginning curvature of the spine to his work of carrying the heavy packages on his left shoulder. At the time of his admission to the clinic in 1896 he had atrophy of the thenar and hypothenar muscles of the left hand, some atrophy of the biceps and triceps of the left arm, and a slight scoliosis with convexity to the left in the

dorsal region. Knee-jerk of the left side was increased; pupils reacted normally. Pain-sense was somewhat diminished in the left arm. Temperature-sense was stated in the history to be probably normal, but he is said to have given contradictory replies in his testing. At that time a loss of faradic excitability of the muscles of the left hand was noted. (Dr. Caswell.)

At present the patient is only fairly well nourished; he is unable to attend to work on account of weakness in the left hand and arm and of a beginning stiffness in the right arm. His scoliosis is well marked, and is evidenced by the photograph.

Atrophies.—Both upper extremities are now affected, though the atrophy in the right side is confined to the hand. On each side there is "main en griffe," though this is less typical in the right upper limb. *Left upper extremity.* Complete atrophy and loss of function and of faradic excitability of the interossei and lumbricales, with little or no galvanic response are noted. The muscles of the thenar eminence are atrophied and have lost their faradic excitability. There is slight response to the galvanic current of 20 to 25 ma., but no qualitative changes on the closure contractions. This same condition is present in the muscles of the hypothenar eminence. The extensor and flexor groups of the forearm are markedly atrophied; the flexors more so than the extensors. None of these muscles have lost their faradic excitability to strong currents, though the reaction is diminished and slower. Reaction to galvanism is much diminished, though not qualitatively altered. The supinator longus acts fairly well. The biceps is in fair condition; the triceps is markedly atrophied in all its divisions. The deltoid, trapezius, supraspinatus, infraspinatus and latissimus dorsi are in fair condition. The serratus magnus is atrophied. The electrical reactions in all of these muscles are similar to those noted in the preceding groups. In the right hand a beginning atrophy of the small muscles is seen. At the present time the faradic excitability has not been entirely lost in the small muscles of the right hand, though the reaction is sluggish and is manifest only with comparatively strong currents. There are no atrophies in the legs and no functional disturbances below the waist that the patient appreciates or that examination can reveal. An apparent slight atrophy of the muscles of the left side of the face is detected, but since the skull is somewhat asymmetric this may be more apparent than real. No functional disturbances of the facial muscles have been observed, save that in January, 1898, there was a diminution of the palpebral fissure of the left side. In January, 1899, this is not so apparent, though still present.

The left pupil is slightly myotic. There is no Argyll-



Figure II. The marks on the arm indicate the areas of analgesia.
Jan. 18, 1898.

Robertson pupil, and in February, 1898, there were no changes in the fundus. No ocular palsies have been seen. Slight lateral nystagmus is found on extreme lateral movements, especially to the right.

Elbow-jerk and wrist-jerk are completely lost. Some fibrillary twitching of a transient character is seen in the muscles of the left arm and forearm. Chronic constipation is marked, and is probably due to bad hygiene, since no loss of sphincter control exists. No vesical disturbances are present, and the sexual appetite is not altered. Knee-jerks are about normal; that of the left side being perhaps increased. Ankle-clonus is not obtained.

Special sensory symptoms consist of a typical dissociation of sensation. No tactile anesthesia is found anywhere in the

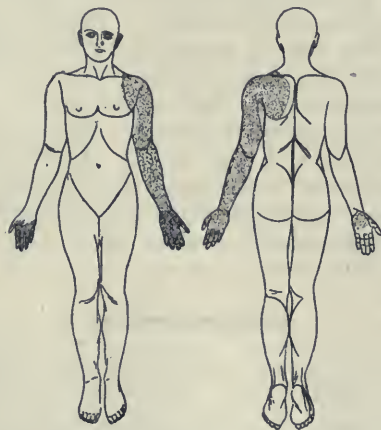


Figure I.—The dotted portions indicate the areas of analgesia and dissociation of sensation; the deeper shading indicates the areas of tactile anesthesia.

body, except in the palms of both hands, as tested with cotton. This does not appear to be unusual in hands which are hardened with hard work, and little stress is, we believe, to be laid upon it. Analgesia to pin pricks, January, 1898, was complete for the whole of the left arm and shoulder, as marked in the photograph taken at the time. In January, 1899, there seems to be a small area sensitive to pain over the biceps muscle. (See Fig. I.) In January, 1898, the dorsum of the right hand was somewhat analgesic, and this analgesia has become more pronounced throughout the year. In February, 1898, a loss of temperature sense was found over the entire left arm, corresponding to the area of analgesia, but recent tests show that the temperature sense has partially returned over the upper parts of this region, and particularly in the palmar surfaces of the hands.

Whether this return is due to actual organic repair or is a result of educational discrimination as a result of many successive testings is not manifest. Cold-conduction is slightly better than heat-conduction. At the present time the palmar surface of the left hand is fairly acute in determining heat and cold when grasping the test-tubes. The percentage of correct answers for this locality being now (January, 1899), 18 out of 20 tests. In 1898 less than 50 per cent. of the determinations were correct. The patient has often burned his left hand with matches, and is himself aware of a diminished heat sensibility. There are no areas of anesthesia or of analgesia in the trunk or lower extremities. The muscular sense (weights) is not impaired, and the sense of position of the hands is within normal range. No changes in gait or in the speech are seen. No Romberg sign, and no girdle sensations exist. Mental changes have not been found. The patient states that his memory and other habits of mind are about as they have been for some time.

Trophic disturbances. Both hands are cold and bluish. At times there is a peculiar velvety edema of a slight grade. Ulcers form readily, and heal slowly. During the summer of 1898 the patient wore a plaster jacket for his scoliosis. As a result, he developed pressure ulcers on his shoulders and hips. These are only now healing after a lapse of four months since the removal of the jacket.

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59. DU ZONA AU COURS DE LA PARALYSIE GÉNÉRALE (Herpes in General Paresis). G. Dupan (Gazette Hebdomaire, 1898, p. 853).

In this observation the patient had an ophthalmic zona at least seven months before the beginning of a general paresis. The disease was distinctly developed in ten months and the autopsy showed that the trigeminus was adherent to the dura mater. Thus it would appear that the eruption was due to an irritation of the trigeminal nerves by the meningeal inflammation.

60. A CASE OF JUVENILE GENERAL PARALYSIS. A. Helen Boyle (Journal of Mental Science, 45, 1899, p. 99).

This patient began to show signs of the disease at about the age of fourteen or soon after. Her family history was obscure. Tuberculosis was present in the family, congenital syphilis could not be ruled out entirely, but its only evidence was an irregularity of child births, the first two children dying soon after birth. The symptoms came on decidedly about the time of menstruation and there would seem to be a distinct relationship with this factor. The chief clinical picture was that of a progressive dementia with no delusions and marked involvement of bladder and rectum. The post mortem examination was held to be confirmatory of the diagnosis.

ANOMALOUS SYMPTOMS FOLLOWING TRAUMATIC HEMORRHAGE INTO THE SPINAL CORD.

LOSS OF KNEE-JERK WITH ANKLE-CLONUS AND GLUTEUS-CLONUS.¹

BY WILLIAM M. LESZYNSKY, M.D.,

CONSULTING NEUROLOGIST TO THE MANHATTAN EYE AND EAR HOSPITAL;
NEUROLOGIST TO THE DEMILT DISPENSARY, NEW YORK CITY, ETC.

Charles N., 35 years of age, born in Sweden, single, carpenter, was first seen by me May 26, 1898. Soon after, he disappeared from observation, but returned January 21, 1899, and gave the following history:

He was in perfect health until June 2, 1896. At that time he fell from the second floor to the basement in an unfinished building and struck on his buttocks. He was unable to get up, as he immediately became paralyzed in both lower extremities, necessitating his removal to the Presbyterian Hospital, where, six hours after the accident, the following condition was noted: "Loss of power in both lower extremities. Sensation lost in the legs to about three inches above the knees; normal in thighs. Areas in leg where tactile sense is preserved, but pain sense is lost. There is an area of anesthesia on right buttock, crescent-shaped, extending about 2½ inches from intergluteal fold. Retention of urine. Constipation." A few days later there was loss of control over the anal sphincter. The patient remained in the surgical ward and gradually improved. He was transferred to the medical division November 16, 1896, where the record shows that there were "paraplegia and some cystitis. Both knee-jerks were diminished and marked ankle-clonus was on both sides." After a sojourn of nine months he was discharged from the hospital, March 10, 1897, nearly two years ago.

He now complains of frequent and almost constant numbness, of a burning sensation in both feet and legs, and of weakness in both lower extremities. He can only walk with the aid of two crutches. He has frequent incontinence of urine, and exaggerated bladder reflex. His bowels are regular and he has good control of the sphincter. Sexual power is not decreased, and he has never suffered from priapism. He sleeps well, and his appetite and digestion are good. No previous history of alcoholism or syphilis.

Status Praesens.—He is a well developed and healthy looking man. The heart, lungs and upper extremities are normal. With slight assistance he is able to stand for a few moments.

¹ The patient and report were presented before the New York Neurological Society, February 7, 1899.

While doing so there is considerable hyper-extension at left knee-joint. In his effort to stand, marked clonus and trepidation of both lower extremities occur. There is incomplete paraplegia with slight spasticity, affecting the adductors of both thighs and the calf muscles. Evidence of local atrophy is absent, but there is general diminution in the consistency and bulk of all muscles, equally distributed in both limbs, as compared with the highly developed and muscular upper extremities.

Measurement.—Circumference of lower extremities, $6\frac{1}{2}$ inches above patella, R. 18 inches, L. 18 inches; $6\frac{1}{2}$ inches below patella, R. $12\frac{3}{4}$ inches, L. $12\frac{3}{4}$ inches.

In the sitting position he is able to fully extend both legs. In recumbency he can elevate the right limb to its full height, while the left can only be raised about three-fourths of the same distance. Both feet are in a condition of talipes equinus, but there is neither ankylosis nor contracture. In the right foot there is only slight power in dorsal flexion. In the left there is none. While lying in the prone position voluntary flexion of legs upon thighs is impossible. There is no evidence of deformity of vertebral column. Muscular resistance to passive movement is quite feeble in the posterior thigh muscles, the crural group and the ilio-psoas on both sides. The power of abduction (gluteus medius and gluteus minimus) is only slightly impaired, while the outward rotation of both thighs (gluteus maximus) is feeble. All of the paretic muscles are flaccid.

Electrical Examination.—Shows quantitative decrease in faradic irritability. Anterior crural nerve and muscles, R. 32 mm., L. 52 mm.; peroneal nerve, R. 25 mm., L. 25 mm.; tibial group of muscles, R. 50 mm., L. 55 mm.; biceps, R. 50 mm., L. 50 mm.; gluteus maximus, R. 50 mm., L. 50 mm. All contractions are slow. The reaction to the galvanic current presents nothing abnormal.

Both plantar reflexes are active. The Achilles-reflex is highly exaggerated on both sides. Ankle-clonus, which is quickly elicited in the usual manner, is equally well marked and persistent on both sides. It is also readily produced by percussion over the Achilles-tendon, with or without dorsal flexion of the foot, and while the patient is kneeling. In fact, the irritability of the gastrocnemius muscle itself is so exalted that a gentle tap upon it will at times produce the same degree of clonus, especially on the left side. This is not influenced in its activity by forced flexion of the great toe.² The left knee-

² From a series of experiments on dogs and rabbits Sternberg has shown (Sitzungs-bericht der Kais. Akademie der Wissensch in Wien, 1891, Vol. C., Part 3) that on percussion over the Achilles-tendon the contraction is not limited to the gastrocnemius, but also includes the soleus and plantaris muscles.

jerk is absent. The right knee-jerk is hardly perceptible, if present at all, with the strongest reinforcement. Percussion just above or below the patellar tendon on either side produces slight contraction of the vastus externus only, but no knee-jerk (except as above mentioned). The contraction of this muscle alone is manifestly too weak to produce extension of the leg, and thus verify the claim of Sherrington³ that the vastus externus is the chief muscle involved in the production of the knee-jerk. Sternberg has also demonstrated that there is no necessary relationship between increase of tendon reflexes and increased muscular tonus, and this latter conclusion would seem to apply in this case, as the mechanical irritability of the vastus externus is increased while the knee-jerk is absent.

From the fact that voluntary extension of the leg upon the thigh is possible, it would seem that the reflex arc is interrupted in its sensory portion within the cord. It is an open question in this particular case as to whether the contraction of the vastus externus is purely reflex or of an idiomuscular type.

Sudden and forcible pressure downward, or percussion below the origin of the gluteus medius muscle on both sides, produces a distinct and persistent clonus which is difficult to suppress, but ceases when the patient forcibly flexes the trunk upon the thighs. The cremasteric reflex is present, but feeble, on both sides. The abdominal reflex is present on the right and absent on the left side. The anal reflex is preserved.

Examination of the urine indicates the presence of a mild degree of cystitis. (Sp. gr., 1029; acid. Excess of phosphates. Muco-pus.)

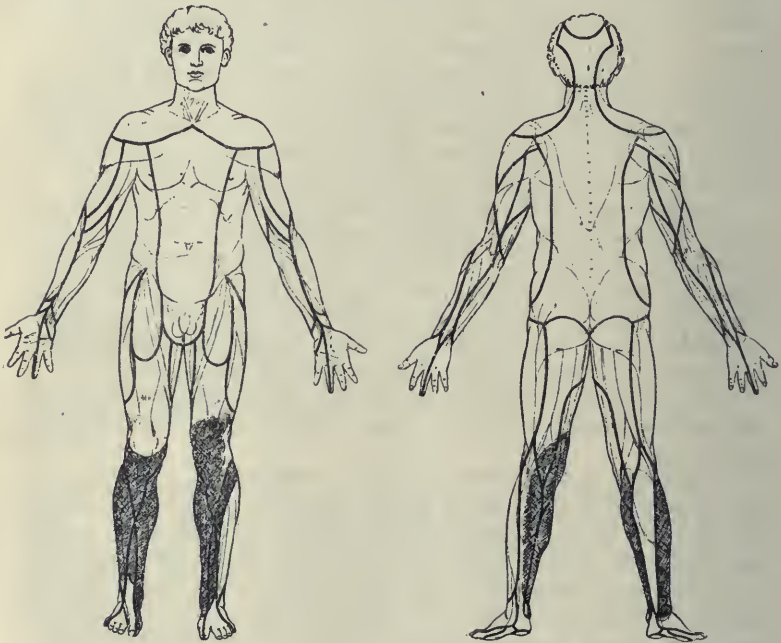
The only objective disturbance of cutaneous sensibility is found almost symmetrically situated in both lower extremities, as outlined in the accompanying chart. In this area tactile sensibility is preserved, while there is complete analgesia and marked impairment of temperature sense. A very strong faradic current with the wire brush applied over the region produces a very slight sensation of pain. The muscular sense is normal in both lower extremities.

It will be observed that while this area of analgesia and thermoanesthesia does not accurately correspond with the picture of sensory disturbance resulting from a complete destruction of the third and fifth lumbar segments, it is sufficiently well defined to enable us to say with some degree of certainty, that the lesion producing it is located in these segments. The dissociation of sensibility, as manifested in the preservation of the tactile sense, and its bilateral and symmetrical distribution, substantiates the conclusion as to its spinal origin.

³ Brit. Med. J., 1892, Vol. I., p. 654.

It is also interesting to note that the area of anesthesia corresponds to the anatomical peripheral distribution of the sciatic (peroneal) and anterior crural (long saphenous) nerves, which innervate respectively the muscles paralyzed in this case, i. e., the biceps and tibial group, and the crural group. From a study of the functions of the various spinal segments, it has been assumed that these muscles are represented in the second and fourth lumbar segments.

The only reflexes that are absent are the knee-jerk and the abdominal reflex on the left side, while both cremasteric



The shaded areas in the lower limbs indicate the regions of preserved tactile sensibility, complete analgesia, and marked impairment of temperature sense.

reflexes are weak. This would indicate involvement of their spinal centers, situated between the two lowest dorsal and the fourth lumbar segments.

The most unusual feature in this case, however, is the absence of the knee-jerk and the presence of ankle-clonus. It seems that this condition occurs in spinal cord lesions limited to the lumbar segments containing the reflex center for the knee-jerk, which at the same time cut off or damage the so-called inhibitory fibers in the motor tract communicating with the lower reflex centers.

If we would accustom ourselves to look upon ankle-clonus as concurrent with an exaggerated Achilles-reflex, and analogous to patella-clonus, which is so frequently associated with a highly exaggerated knee-jerk, the anomalous condition of ankle-clonus with loss of knee-jerk would become more comprehensible.

From a study of 1900 cases observed in the psychiatric clinic at Jena, Ziehen* found that excessive increase of the Achilles-reflex without foot-clonus was exceedingly rare. Foot-clonus without distinct increase in the Achilles-reflex he observed only twice. All of the other reflexes in my case whose spinal centers are in the sacral portion of the cord are either active or exaggerated.

The pronounced and excessive clonus affecting the deeper gluteal muscles is a condition that is rarely observed, and probably indicates an irritative character of the cord lesion, as well as damage to the inhibitory fibers.

It is a common experience to note the loss or diminution of sexual power in traumatic lesions affecting the lower portion of the cord. The preservation of function in this case may be explained by the escape from damage of the genital centers in the sacral segments. From the foregoing, we may conclude that primarily, there was a traumatic hemorrhage into the lumbar cord, with irregular disintegration of structure in the upper lumbar segments. This has probably resulted in a secondary degeneration affecting symmetrically the motor tract below this area, and in the formation of a cavity in this region. The lesion evidently extends irregularly from about the tenth dorsal to the upper sacral segments.

* Ziehen, *Deutsch. med. Woch.*, 1894, Nos. 33 and 34.

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61. UEBER PRURITUS ALS SYMPTOM DER PROGRESSIVEN PARALYSE (Pruritus as a Symptom of General Paresis). A. Sarbo (*Pester med. chirurg. Presse*, 33, 1897).

In two cases the author observed a persistent and intense pruritus as an early symptom of general paresis. This symptom disappeared as the disease progressed.

62. BEITRAG ZUR ÄTIOLOGIE DER PROGRESSIVEN PARALYSE (Contribution to the Etiology of General Paresis). W. Eccard (*Ver. Bl. der pflz. Ärzte*, 13, 1897, No. 11).

The author believes that without syphilis there can be no general paresis. In a study of 35 cases, 24 men and 11 women, in the men a positive history was obtained for 22, 91.9 per cent., in the women, 8 cases had been infected, 72 per cent.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

February 7, 1899.

Vice-President, Dr. Joseph Collins, in the chair.

CASES OF PARTIAL SPINAL CORD INJURY.

Dr. Pearce Bailey presented two cases of this kind. The first was that of a man who on December 9, 1898, fell several feet, struck on his head, and became unconscious. On recovering consciousness he had tingling in both upper and lower extremities, but no disturbances of the bladder or rectum, and no real paralysis. He soon afterward left the hospital, but since then had been troubled with pain and stiffness in the neck, and with numbness in the extremities on the right side. Examination at the time of presentation showed anesthesia of the right forearm and right leg, more marked in the latter, and a difference in the palpebral fissures. The left pupil was the smaller and less responsive. No clonus, no paralysis and no twitchings were observed. A distinct prominence was seen in the cervical region, but the stiffness of the neck was rapidly diminishing. The case was looked upon as one of slight unilateral hemorrhage in the sixth cervical segment, with possibly some contusion.

The second case was that of a man, forty-three years of age, who had been squeezed between two heavy masses in an elevator shaft last November, and while thus caught the elevator had descended upon his head, and pressed him down to the floor. He had not regained consciousness until after reaching the hospital. He had then severe pain in the neck and tingling in the fingers, but had control over the bladder and rectum. Examination at the time of presentation showed anesthesia over the right deltoid region, and exaggeration of all the reflexes, but the right patellar reflex was not as lively as that on the left side and a left foot-clonus had been noted. The case was an incomplete type of Brown-Séquard's paralysis. In view of the forcible flexion of the neck it was almost certain that a hemorrhage into the cord had occurred.

Dr. Edward D. Fisher said that he had under observation a case in which his diagnosis had been meningeal hemorrhage, and possibly a slight hemorrhage into the spinal cord, but because of the history

he could not feel that a very serious loss of substance in the spinal cord had occurred. The condition of the reflexes seemed to indicate the existence of some compression outside of the cord, either the result of hemorrhage or of slight dislocation of the vertebræ. Three months had elapsed since the accident, yet the boy was only just able to move around. Unless paralyses were associated with atrophy he did not feel sure of the existence of a large hemorrhage in the cord.

Dr. George W. Jacoby believed that cases of this kind were examples of subarachnoid hemorrhage, and that the diagnosis could be made by lumbar puncture. If pure fluid, free from admixture with blood, were drawn off, there was evidently no subarachnoid hemorrhage. He also thought that this procedure possessed some therapeutic value.

Dr. Bailey stated that the diagnosis in his cases had been based mainly on collateral evidence. The records of autopsies showed that extradural hemorrhages were extremely rare, except when associated with mutilation of the cord. In the examinations that he had made on spinal cords of several persons who had met violent death from general traumatism, he had found small hemorrhages into the cord without any fracture of the vertebræ. This was particularly true of injuries in the cervical region.

Dr. W. M. Leszynsky presented a case of spinal cord lesion with anomalous symptoms. (See p. 231.)

DEMONSTRATION OF THE BRAIN OF A CASE OF SENSORY APHASIA.

Dr. Joseph Collins reported the case of a negress, who had sensory aphasia. The patient when first seen had hemiplegia, not entirely typical, and at intervals of eight months had had attacks of epilepsy. The most striking defects of spontaneous speech were amnesia of words, particularly of nouns, and paraphasia. She could usually tell her name, but not the names of members of her family or her residence. It was impossible for her to repeat the simplest sentence after it had been spoken. She could sing the airs of popular tunes, but could not get the words in right. She had lost the comprehension of written and printed words. After repeated examinations right lateral hemianopsia had been demonstrated. She had had a slight degree of word-deafness, which became more apparent the longer the case was studied; it was due apparently to functional word degradation. A lesion of the visual center and subjacent white substance was believed to be present. If she were asked to say "B," she might say "C" instead, and in other ways she presented many instructive contrasts in connection with her aphasia. She died about two months ago.

On post-mortem examination, the brain was extremely small, and the left hemisphere was distinctly smaller than the right. There were also three areas of softening in the left hemisphere—one involving the middle third of the ascending parietal convolution; a second area situated in the posterior end of the inferior parietal convolution, and involving the entire

angular gyrus, except its inferior portion, and a third area in the posterior end of the supratemporal convolution. The first area of softening measured 3 cm. perpendicularly, and 1.6 cm. at the widest part. The second area was the largest, measuring in its antero-posterior diameter 2 cm., and 1.5 cm. vertically. The most conspicuous alteration of the superior temporal convolution was its shrinkage, so that, as compared with the other side, it looked like a ribbon. The pia was not adherent except at one portion on the left side, and over the posterior end of the fissure of Sylvius. When the pia was stripped from the fissure of Sylvius, the destruction of tissue in the areas referred to was seen to be greater than had appeared at first sight. The cuneus had remained intact. The base of the brain showed no abnormalities, except the relative smallness of the left hemisphere. The optic nerve and chiasm were normal, the blood vessels were not thickened, and there was no evidence of meningeal inflammation. The medulla oblongata and cerebellum were normal in appearance and relationship. These findings seemed to him exceedingly interesting in view of the clinical history and the diagnosis made during life. He expected, on dividing the brain, to find that the optic radiations had been severed, thus giving rise to the homonymous hemianopsia.

Dr. Fisher expressed his surprise that in a case lasting only three years there should have been such a marked difference in the two hemispheres.

Dr. William Hirsch read a paper on the relation of infantile spinal paralysis to spinal diseases of later life. (To be published in this journal.)

Dr. Edward D. Fisher agreed with the reader of the paper that the case was not one of amyotrophic lateral sclerosis. The point had been very clearly brought out that many cases formerly diagnosed as systemic diseases were really various forms of diffuse myelitis. He said that many cases of poliomyelitis were met with which extended over a long period, and yet they did not give rise to the condition found in the case just reported; hence, the direct relation of the inflammatory condition to the original poliomyelitis could not be definitely established. The theory of Strümpell that a virus could remain latent for many years and then exert its influence seemed to him very far-fetched. He would like to ask whether there was any direct connection between the bulbar symptoms and the spinal symptoms which were present primarily. The clinical picture of poliomyelitis was almost perfect, so that no other diagnosis could be expected under the circumstances.

Dr. Leszynsky said that he had had under observation for eight years a lady now of forty-eight years, who during her childhood had had a poliomyelitis. This had left her with an incompletely paralyzed limb. Some years later, she had developed loss of power in both lower extremities, and when first seen by him some years ago all of the signs of a progressive chronic poliomyelitis in both lower extremities were present. The disease had progressed, although very slowly, since that time. This seemed to indicate the possibility of a

direct relationship between the old lesion and the later one, but this was the only case of the kind that he had ever encountered.

Dr. George W. Jacoby asked if cases of encephalitis occurred frequently or at all in later life after cerebral hemorrhage in childhood. He had seen one such case, in which an encephalitis occurred in an adult who had had a cerebral hemorrhage in childhood. There seemed to be an analogy between this and the case reported in the paper.

Dr. Joseph Collins did not think that there was any relation between the poliomyelitis and the diffuse myelitis occurring later in life in Dr. Hirsch's case. He could not imagine how the previous scar could have anything to do with starting the later inflammation from which the patient died. According to clinical experience, with the bare exception of keloid, scars were not the starting-point of such pathological processes. The whole thing seemed to him a mere coincidence. In spite of the great care exercised in the examination he had not been convinced that any definite relation between the two diseases had been demonstrated. It was naturally very gratifying to him that Dr. Hirsch's study had confirmed his own observations regarding the allocation of function of the posterior group of cells in the eighth dorsal segment. The explanation offered by the reader of the paper for the absence of changes in the Rolandic cortex also seemed very plausible and trustworthy. He certainly agreed with Dr. Fisher that it was ridiculous to suppose that a scar could lock up a poison for half a lifetime, and then allow it to come out and produce disease.

Dr. Hirsch closed the discussion. Regarding the connection of the bulbar symptoms and the spinal cord symptoms, and the relation between the infantile spinal paralysis and the later disease, he said that he had quoted theories largely, but it seemed to be an actual fact that the pathologic process started from where the so-called scar of the poliomyelitis had been. This had been proved both by the clinical symptoms and the pathological findings. The fan-like proliferation of connective tissue into the pyramidal tract accounted for the rigidity of the limbs at the beginning of the later disease. The order of development of the symptoms seemed to point to the origin of the later disease from the left anterior horn. Strümpell had compared the "scar" to a tubercular focus—evidently not an absurd comparison. He had found the report of a case of infantile poliomyelitis in which encephalitis occurred in later life.

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63. FO FILFÁLDE AF LANDRY'S PARALYSE (Two Cases of Landry's Paralysis). A. Schultz (Nordsk. Mag. f. Laegevidensk, 59, 1898, No. 6).

One of the cases here reported is of interest in that it presented all of the clinical features of an acute ascending neuritis, resulting in death by paralysis of respiration at the end of two weeks, and yet the microscopical findings, by modern methods, were entirely negative. The second case followed a multiple neuritis in a chronic alcoholic patient. Here there was a diffuse parenchymatous myelitis and a parenchymatous degeneration of the nerves.

JELLIFFE.

PHILADELPHIA NEUROLOGICAL SOCIETY.

January 23, 1899.

The President, Dr. F. X. Dercum, in the chair.

Dr. F. A. Packard read a paper with the title "Acute Anterior Poliomyelitis Occurring Simultaneously in a Brother and Sister." (See p. 210.)

Dr. Charles K. Mills said that in recent years he had come to the conclusion that anterior poliomyelitis is an infectious disease, but that he had very little experience with its occurrence in members of the same family. The fact that the disease frequently occurs in healthy children with a good family history would seem to lend strength to the view that it is of infectious origin.

Dr. Charles W. Burr remarked that a disease occurs in pigs which probably is an anterior poliomyelitis. The disease begins with slight fever, the animal appears to be sick, then it becomes palsied, and later the muscles waste. He knew nothing as to the condition of the reflexes. Sometimes the hind legs are affected, sometimes four legs and sometimes three. Sensory symptoms seem to be absent. If the pig is pinched, it squeals. Sometimes all the pigs in a pen are affected, sometimes only one is attacked. This disease is certainly infectious and he thought that it was the same as that which occurs in children.

Dr. F. X. Dercum presented a case of chronic poliomyelitis of the adult, affecting the entire cord and involving also the medulla.

The patient was a farmer, 69 years of age, who one year previously had noticed occasional huskiness in his voice. This huskiness had gradually increased and finally led to an examination of the throat by a laryngologist, who stated that the patient presented a paresis of the vocal cords. The patient was then referred to the nervous clinic of the Jefferson Hospital on December 12, 1898. The symptoms of bulbar palsy were at once noted. Phonation was extremely difficult. There was marked paralysis with atrophy of the tongue and marked inability to move the lips, especially to imitate the movements of pouting.

On stripping the patient, marked muscular wasting was found in the arms and legs of both sides and also in the trunk. The atrophy was most marked in the muscles of the shoulder girdle; the deltoids and supraspinatus and infraspinatus muscles showing the greatest degree of change. Fibrillary tremors were widely diffused and noticeable not only in the muscles in which atrophy had become pronounced but also in

numerous other groups. Distinct atrophy was also present in the gluteal regions, thighs and legs. Fibrillary tremors were also noted in these situations. The knee-jerks and elbow-jerks were distinctly minus. No sensory phenomena were observed, nor were there any ocular manifestations.

Dr. Dercum stated that in his experience cases of poliomyelitis in the adult, so extensively diffused, were comparatively rare. The more common syndrome observed is that in which atrophy is first manifested in the hands, in the thenar and hypothenar eminences, and as the case progresses, spastic symptoms make their appearance in the legs; the legs not showing atrophy as do the arms, but on the contrary presenting more or less rigidity with exaggeration of the knee-jerks. In other words the syndrome of amyotrophic lateral sclerosis, with or without involvement of the medulla, is the more common form of muscular atrophy of spinal origin in the adult.

Dr. Dercum also placed on record a case of organic hemiplegia with hemianesthesia, extending over five years.

J. B., male, aged 49; a mulatto; an inmate of the wards of the Philadelphia Hospital, had suffered from an apoplectic seizure five years ago, the attack being followed by hemiplegia of the left arm and leg, together with a sharply defined and persistent hemianesthesia of the same side. The man presents to-day signs of a spastic hemiplegia of the left side, moderate in severity, together with a moderate degree of contracture of the left arm and spastic rigidity and exaggeration of the knee-jerk of the left leg. The hemianesthesia at present is sharply delimited by the middle line of the body and appears to be everywhere complete. A right sided homonymous hemianopsia is associated with this hemianesthesia. There is no impairment of hearing upon the left side nor is there any involvement of taste or smell. The sensory symptoms are limited to those concerned in the various forms of cutaneous sensibility and the visual phenomena just stated. There can be no doubt that in the present instance the lesion was so situated as to involve not only the fibers of the anterior two-thirds of the posterior limb of the internal capsule, but also the posterior third of the posterior limb and the fibers of the optic radiations. Cases of hemiplegia, even of organic origin, with *persistent* hemianesthesia are in the experience of Dr. Dercum comparatively rare. Hemianesthesia and hemianopsia are not infrequently observed during an apoplectic seizure, or immediately subsequent to a seizure, but it is quite common for these symptoms to disappear at a relatively early period, and they are therefore to be regarded as distance symptoms. Actual lesions of the posterior portion of the internal capsule and of the optic radiations must be a rather rare occurrence.

Dr. W. G. Spiller spoke of the recent views regarding the location of the sensory fibers. It had been believed that the external bundle of the peduncle was sensory, because it did not degenerate downward after lesions of the inner capsule. Dejerine had shown by several cases that this view was incorrect, and that this bundle of fibers has its origin in the second and third temporal convolutions, and does degenerate downward if the lesion in the internal capsule extends downward sufficiently to cut these fibers. Mills and Spiller had confirmed the observations of Dejerine, to the extent of showing that the external bundle of the cerebral peduncle does not arise in the first temporal convolution, and Spiller had published the report of another case establishing the same fact. The case of Mills and Spiller was one of abscess in the first temporal convolution with absence of all degeneration in the external bundle of the peduncle by Marchi's method. The case of Spiller was one of infantile hemiplegia of many years duration, with destruction of the first temporal convolution and integrity of the external bundle of the peduncle.

Dr. Spiller referred to the views of Dejerine regarding the absence of a distinct band of fibers for the conduction of general sensation in the area posterior to the posterior limb of the internal capsule. He stated that Dejerine and Long had observed two cases in which a lesion was found in the posterior limb of the internal capsule, the thalamus was intact and sensation, therefore, had not been altered.

Dr. Charles K. Mills thought that it was not unusual, although not common, to find persistent hemianesthesia with hemiplegia in organic cases. He had seen a number of such cases. One of these was a patient who had been under observation for many years in the Philadelphia Hospital. The case was interesting in that it presented hemiplegia and hemianesthesia without hemianopsia. The case however throws no positive light upon the part played by lesions of the internal capsule in the causation of hemianesthesia, as the lesion involved both the thalamus and the posterior part of the internal capsule. The anesthesia was probably due to the lesion of the thalamus. He thought it probable that in Dr. Dercum's case a lesion involved both the posterior part of the capsule and the thalamus, and that perhaps the hemiplegia was due to the lesion of the capsule and the anesthesia to the lesion of the thalamus. He thought that the question of the passage of sensory fibers through the posterior extremity of the posterior limb of the internal capsule was not yet settled, as some physiological and pathological observations seemed to support the older view of the sensory functions of a portion of the capsule.

A CASE OF ANOMALOUS DYSTROPHY.

Dr. S. Solis Cohen presented a woman, 48 years of age, with a peculiar enlargement of the hands and feet. The menopause had occurred 6 years ago. The hands had always been rather large, but during the past three months they had steadily increased in size, and at the time the patient was presented were much larger than they had been a year ago. They were broadened and deflected to the ulnar side, and the fingers presented a conical outline rather than the sausage-shape of acromegaly. The feet had also increased in size. Skiagraphs showed that the enlargement of the hands was due to hyper-

trophy of the soft tissues, and not of bone. Some wasting was noticed in the first interosseous space. The nails were normal. The grip of the hands was weak. The forearms were normal, but pads apparently of fat were found on the arms. Paresthesia was felt in the hands and feet only during wet weather.

The feet were symmetrically enlarged, except that the increase of size was more noticeable over the malleoli. The enlargement was confined to the soft tissues.

The patellar reflex was much exaggerated, but ankle-clonus was not obtained. All forms of sensation were normal. Slight kyphosis was observed in the cervical region. Headache was not constant. The bones of the face were not enlarged. Over the lower jaw was a freely movable fatty mass giving a pointed appearance to the chin. Hemianopsia was not observed.

Dr. Guy Hinsdale agreed with Dr. Burr and Dr. Cohen that the case did not present the physiognomy of acromegaly. The absence of enlarged and prognathous jaw, the character of the fields of vision, the absence of exophthalmos, the absence of disease of the thyroid, the absence of history of disturbance of the menstrual function and the appearance shown by the skiagraphs would indicate a disease not related to acromegaly. He thought that the history of two attacks of typhoid fever was of interest as it is well known that the toxins produced in typhoid fever may cause remarkable changes in nutrition. Possibly this might prove to be the ultimate cause of the trophic changes in the extremities.

Dr. A. A. Eshner referred to the possibility of this being some form of adiposis or lipomatosis. As the skiagraphs showed and as physical examination indicated, the changes were confined to the soft tissues. It seemed to him that there might be a series of dystrophies with myxedema and cretinism at the one end and, perhaps, acromegaly at the other, not necessarily related. Whether or not simple obesity is related to this group of diseases is of interest. With regard to the apparent condition of the thyroid, he did not consider that a matter of much importance, for it is possible for the thyroid to be outwardly and grossly normal, while its function is seriously deranged.

Dr. David Riesman was reminded by this case of one reported recently by Lunz—the case of a woman with syringomyelia, who had marked enlargement of the right upper extremity, and a hand much like those of Dr. Cohen's patient. Lunz, following Marie, called the hypertrophy cheiromegaly. Dr. Riesman believed Schlesinger had reported a similar condition in syringomyelia under the name of macrosomia. He was unable to say whether Lunz's case threw any light on the case of Dr. Cohen.

Dr. William G. Spiller said that distinct atrophy of certain of the interossei muscles in one of the patient's hands had been noticed. Muscular atrophy is common in joint disease, and Dr. Cohen had obtained crepitation in some of the joints of the hand. It had occurred to Dr. Spiller that perhaps the hands in this patient corresponded to some extent to the *main succulent* described by Marinesco as pathognomonic of syringomyelia. Several cases have shown that this condition is not pathognomonic of syringomyelia and that it may be found in other diseases.

Dr. Wharton Sinkler thought that the hands of this patient were strikingly like the hands of a patient at the Philadelphia Hospital who

had syringomyelia. The question arises, however, can we conceive of syringomyelia without any sensory changes whatever.

Dr. Spiller remarked that a case of syringomyelia without sensory disturbance and with necropsy had been reported by Dejerine and Thomas.

Drs. Musser and Sailer reported 8 cases of meralgia paræsthetica; 4 in males and 4 in females. The apparent causes of the condition were: pressure of an army belt followed by severe infectious disease; a neuropathic tendency, with history of a similar condition in the grandfather; neurasthenia; pregnancy in 2 cases; influenza; and vesicular eczema. One of the cases occurred in a woman suffering from neurasthenia who seemed to be worse during the menstrual period.

64. POLYNÉVRITE OURLIENNE AU COURS DE LA GROSSESSE (Multiple Neuritis from Mumps Occurring During Pregnancy). Louis Gallavardin (Lyon Médical, 89, 1898, p. 97).

Paralysis occurring in the course of or following mumps may be divided into three categories:

1. Facial paralysis caused by the direct invasion of the facial trunk by the parotitis.
2. Paralysis of central origin, cerebral or meningeal, of which Lannois and Lemoine have reported four examples. To these Glénérat and Liégeois have each added one case.
3. Paralysis due to peripheral neuritis such as may be caused by any infectious disease. It is an example of this last class that the author reports.

A woman of thirty years, seven and a half months pregnant, was perfectly well up to three weeks before admission to hospital when she was taken with mumps affecting both parotids. The swelling of these glands had disappeared at the end of a week when she began to suffer from pruritis of the entire body, but especially of the extremities, which lasted four or five days and was succeeded by pains in arms and legs. Soon thereafter general weakness made its appearance and increased until she was bedfast.

Examination revealed paresis, almost amounting to paralysis, affecting the entire body, loss of muscular sense and diminution of cutaneous sensibility increasing from the trunk towards the ends of the extremities. Knee-jerk and plantar reflex were abolished, the sphincters unaffected and mentality normal.

The patient grew worse for the next two weeks when labor came on and she was delivered of a living child, at the end of the eighth month. After the confinement, improvement was gradual, but practically continuous, and two months later muscular power was very good, but co-ordination was so bad that she was absolutely unable to walk. Nearly three months later there still remained marked ataxia of arms and legs and the knee-jerks were absent. PATRICK.

Periscope.

65. UEBER DIE LEITUNGSBAHNEN UND PATHOGENESE DER RINDENEPILEPSIE (Concerning the Conducting Tracts and the Pathogenesis of Cortical Epilepsy). Johann Prus (Wienerklin. Wochenschrift, 38, 1898, p. 857).

Prus has made over a hundred experiments on dogs to determine the nature of cortical epilepsy.

The cortical epilepsy does not become generalized by the irritation extending throughout the cerebral cortex, similar to the formation of concentric waves following the falling of a stone into water, as Unverricht believed; and Unverricht's "irradiation law" is incorrect. When the cortical centers of certain muscles were extirpated, these muscles participated in the general convulsions after other parts of the cortex were strongly irritated, and removal of almost an entire hemisphere did not prevent bilateral convulsions. When a glass tube of about 2 cm. in diameter was driven into the motor area of the brain, and the cortex so enclosed was irritated, a general convulsion followed, although the irritation could not be transmitted through the glass wall.

Prus' experiments seem to prove that division of both pyramidal tracts in dogs, at any level, does not prevent general convulsions from irritation of either cerebral hemisphere. The pyramidal tracts, therefore, have no part in the generalization of the cortical epilepsy.

Prus found that when he cut through the midbrain at the level of the posterior quadrigeminal bodies, without involving the pyramidal and pontile tracts in the crusta, he could not produce epileptic attacks, even by the strongest irritation of the motor cortex, although he could produce spasm of the muscles whose centers were irritated, but only during the time the electrode was applied. The tracts, therefore, which convey the irritation necessary for the production of epileptic convulsions are not the pyramidal, or the other tracts in the crusta, but are "extrapyramidal" tracts, and they pass through the tegmentum or substantia nigra of the mesencephalon.

Prus found that electrical irritation of the motor cortex, after it had been painted with a ten per cent. solution of cocaine, did not produce epileptic convulsions, although it did cause spasm of the muscles represented in this area, and of all other muscles of the body when the current was sufficiently strong; these spasms ceased, however, as soon as the electrode was removed. No loss of consciousness was produced under those conditions. Bilateral epileptic convulsions could be obtained when the electrical irritation was applied to the motor area of the hemisphere which had not been painted with cocaine, provided the attempt was made soon after the application of the drug. The anesthetic applied to one hemisphere affected both after a time, and epileptic convulsions could not be obtained by irritation of either hemisphere. These experiments, Prus thinks, prove that sensory fibers, especially their end ramifications, are present in the motor areas, and that the end ramifications of the sensory fibers or the sensory ganglion cells of the cortex—and not the motor cells or the motor fibers of the cortex—give origin to the cortical epilepsy.

Motor "extrapyramidal" tracts undoubtedly exist. Prus cut com-

pletely both pyramidal tracts in the anterior pyramids, or in the cerebral peduncles—in the latter case cutting also the so-called pontile tracts of the crusta—after he had anesthetized the motor cortex, and he then obtained contraction of the muscles by cortical irritation with the faradic current. He believes he has demonstrated the existence of motor “extrapyramidal” tracts and of their decussation in the medulla oblongata, because he prevented all reflex action by the anesthetization of the cortex, and because the muscular contractions could not have been caused by way of the pyramidal or pontile tracts. He believes also that the “extrapyramidal” tracts are for associated movements; for example, he obtained extension of the foreleg on the side opposite to, and of the fore and hind legs, on the same side as the irritated anesthetic cortical area, after he had cut both pyramidal tracts. This he regards as an associated movement.

Prus found also that when he cut one-half of the midbrain just behind the posterior quadrigeminal bodies, and then irritated the cerebral cortex of the same side, he was able to obtain bilateral epileptic attacks. When he further cut one-half of the lower portion of the medulla oblongata, or of the spinal cord, on the side opposite to the injured half of the midbrain, he was still able to obtain bilateral epileptic convulsions from cortical irritation. These experiments showed that the epileptic attacks became general by means of the gray matter of the midbrain, medulla oblongata and spinal cord.

There is no special epileptic center, and there is no essential difference between cortical and “genuine” epilepsy. SPILLER.

66. *UNTERSUCHUNGEN UBER DIE FEINERE STRUCTUR DER NERVENZELLEN UND IHRER FORTSÄTZE* (Investigations of the Finer Structure of the Nerve Cell and its Processes). Vladislav Ruzicka (Archiv. f. Mikroskopische Anatomie und Entwicklungsgeschichte, 53, 1898, p. 485.)

The author presents the following conclusions:

- (1.) The Nissl bodies are not preformed in the living nerve cell. They are artefacts, not produced, as maintained by Held, by acid fixatives, but by the process of decolorization.
- (2.) The anterior horn motor cells of the ox and dog do not possess throughout, a parallel striped protoplasmic structure. This arrangement, when it exists, cannot be held to be characteristic for a motor-acting cell.
- (3.) The nerve cells of the spinal cord may be united to one another by anastomoses.
- (4.) The nerve cells of the spinal cord send out fine processes from their surfaces which pass into the surrounding tissues.
- (5.) The larger dendrites as well as the finer processes of the nerve cell probably act in some anabolic capacity. JELLIFFE.

67. *BEITRÄGE ZUR HISTOPATHOGENESE DER TABISCHEN HINTERSTRANGDEGENERATION* (Contributions to the Histopathogeny of the Tabetic Degeneration of the Posterior Columns.) Karl Schaffer (Deutsche Zeitschrift für Nervenheilkunde, 13, 1898, p. 287).

Schaffer says that the cases of incipient tabes, especially the cases of general paralysis with involvement of the posterior columns, show a degeneration of areas exactly corresponding to those which have been observed in the fetus, and he believes that the tabetic degeneration is confined in its early stages to the embryonal fiber systems of Flechsig and Trepinsky. The study of degeneration of isolated pos-

terior roots shows that a single posterior root contains fibers belonging to different embryonal systems, and that the embryonal systems are composed of posterior root fibers. As the degeneration of incipient tabes corresponds with embryonal zones, it follows that in tabes the posterior roots do not degenerate in toto, but that certain fibers of each affected root degenerate, and that these affected fibers correspond to the fetal fiber systems.

Schaffer believes that the ventral fields of the posterior columns contain exogenous as well as endogenous fibers, and that the same is probably true of Gombault and Philippe's triangular zone. Tabes is a systemic disease of the posterior columns, and this posterior column affection is secondary to degeneration of the posterior roots. The degeneration of tabes is systemic and segmental; i. e., it follows the embryonic divisions of the posterior columns, and is in the intra-medullary distribution of each altered root.

Schaffer has shown elsewhere that the spinal ganglion cells are not altered in tabes, even when they are studied by the method of Nissl, and this fact he regards as a proof that the primary lesion of tabes is not in the cells of the spinal ganglia. The degeneration of the posterior roots in tabes may not always be elective, but a root may degenerate in toto, and most frequently both forms of degeneration are found in the same case. Infiltrative and vascular lesions of the posterior roots, producing nutritive and circulatory disturbances, play an important role in the causation of tabes; but a primary degeneration of the posterior roots may also occur. The intraspinal portion of the posterior root is more vulnerable than the extraspinal, and the degeneration begins in the former portion. The point of contraction in the posterior root (Obersteiner and Redlich's *locus minoris resistentiae*) is not always the part surrounded by the pia. It does not follow that a posterior root degenerates in toto, because it has been injured at a certain point. Schaffer has shown that the different tracts of the spinal cord degenerate at different periods, when the cord has been cut transversely; and it is probable that an entire posterior root may be exposed to injurious agents, and yet only certain fibers belonging to definite tracts may degenerate, or at least, degenerate within a certain time. In this way the tabetic degeneration may conform to the embryonal systems of the posterior columns, even though the whole of the posterior root may be exposed to injury.

SPILLER.

68. BEITRAG ZUR KENTNISS DER „SECUNDÄR MALIGNEN NEUROME.“
(Contribution to the Knowledge of Secondary Malignant Neuroma).
Habermann, (Münchener Medicinische Wochenschrift, 23 and 24,
1898).

Multiple neuromata seem to bear a close relationship to multiple fibromata of the skin. Their development produces the clinical picture of a rare disease, "neurofibromatosis" or "elephantiasis nervosum," which is apparently always congenital in origin. These neurofibromata seem to have a special tendency to undergo sarcomatous degeneration, though they may exist for years without giving any symptoms before this begins. A distinction is to be drawn between secondary malignant neuromata, and primary sarcomata of nerves.

In the first, hereditary predisposition is usually made evident by the presence of multiple fibromata, and often of some defect or deformity. In primary sarcoma these elements are wanting. Primary sarcoma soon breaks through its capsule, and forms metastases. Secondary malignant neuroma grows rapidly, but generally remains enclosed in its capsule, and has little tendency to form metastases. The rarity of

secondary malignant neuroma is shown by the fact that only twenty-two cases are recorded in literature.

To these the author adds a twenty-third, of whose history the following is a synopsis:

The patient, a thin, dwarfish woman of twenty-eight, had had since childhood, a dorsal scoliosis, and did not menstruate until twenty-four years old. Over her body she had a diffuse brownish pigmentation, with numerous nevi-like spots, and on arms, breast, abdomen, and left leg fibromata of different sizes and consistencies, some pigmented, others not. In the spring of 1893 she experienced pain of a severe and burning character in the right foot, and on the right buttock there developed a swelling of great sensitiveness. Upon examining her in November, 1893, the author found here a very painful tumor of the size of a duck's egg. This was connected with the sciatic nerve. This he removed, having to cut out a piece of the nerve, and to join the ends by a procedure analogous to the tendon-splitting of Hueter and Czerny.

The patient made a good recovery, with gradual return of sensation and motor power. The tumor proved to be a fibrosarcoma arising from the endoneurium, and contained a few nerve fibers.

The patient soon commenced to feel pain and weakness in the right arm, and in February, 1895, a small tumor connected with the ulnar nerve was removed with good result. This proved also to be a fibrosarcoma.

Nine months later, after the same set of symptoms two small beginning fibrosarcomata were removed from the left occipital nerve. The patient next developed severe pain in the left hypogastric region, the sensitiveness and tension of the abdominal muscles being so great that no satisfactory exploration of the abdomen could be made. After lasting for four months, during which time morphine was constantly necessary, for some unexplained reason the pain suddenly diminished, and the patient was able to get about again. The trouble still progressed, however, and in November, 1896, there were extirpated the following tumors:

Two on the head (plexiform neuroma), one on the thorax, two on the buttocks, two on the thigh, one in the radial region. All were connected with nerves. The case has continued to progress in the same way, more tumors constantly developing, though by the use of morphine in small doses, the patient keeps about, and does a little housework. Some of the latterly extirpated tumors have proved to be pure neurofibromata. The microscopical report is given in full, and the subject in its pathological aspect is discussed at length. ALLEN.

69. UEBER VERÄNDERUNGEN DER SPINALGANGLIENZELLEN UND IHRER CENTRALEN FORTSÄTZE NACH DURCHSCHNEIDUNG DER ZUGEHÖRIGEN PERIPHEREN NERVEN (Concerning Changes in the Cells of the Spinal Ganglia and their Central Processes after Division of the Peripheral Nerves). R. Cassirer (*Deutsche Zeitschrift für Nervenheilkunde*, 14, 1898, p. 150).

Cassirer removed several centimeters of the sciatic nerve in sixteen rabbits, near the sciatic foramen, and examined, at various periods following the operation, the spinal ganglia belonging to these nerves. He distinguishes four types of normal cells in the spinal ganglia of rabbits. He believes with v. Lenhossék that a pericellular space does not exist when the technique has been perfect. He was able to observe pathological changes in the cells of the ganglia as early as five days after the operation. After about fifteen days the

normal cells began to be more numerous than the pathological ones. The alterations he observed were enlargement of the Nissl bodies; peripheral and, later, complete chromatolysis; displacement of the nucleus; possibly diminution in the size of the nucleus; greater staining properties of the nucleus; and—when the degeneration was advanced—pale blue coloration of the protoplasm. He found also some degeneration of the posterior columns and posterior roots on the operated side, and in the region corresponding to the sciatic nerve. It may be stated, as a result of his experiments, that when the sciatic nerve in the rabbit is cut, many cells of the corresponding ganglia are altered, and most of these are later restored. The escape of certain cells, and the restoration of others, cannot be satisfactorily explained. While changes were found five days after the nerve was cut, degeneration of the posterior columns was not found until fifteen days after the operation; this would seem to indicate that the latter was the result of the former. The degeneration of the posterior columns was slight, and the spinal-ganglion cells which showed the highest degree of alteration were not numerous. Cassirer thinks the results of these experiments do not offer an explanation for the degeneration of tabes.

SPILLER.

70. EIN BEITRAG ZUR PATHOLOGISCHEN ANATOMIE DER EPILEPSIE (A Contribution to the Pathological Anatomy of Epilepsy). A. Alzheimer (Monatsschrift für Psychiatrie und Neurologie, vol. iv, 1898, p. 345).

Alzheimer describes two cases of epilepsy with dementia, in which he found degeneration of the medullated fibers of the cerebral cortex, especially of the tangential layer, proliferation of the glia, and destruction of ganglion cells. These changes were almost uniform throughout the cerebral cortex, but the vessels presented no important alteration. He believes from his investigations and those of others that cases of so-called genuine epilepsy exist, with characteristic pathological changes. In these cases there is macroscopically more or less sclerosis of the superficial cortical layer, and microscopically proliferation of the glia with a tendency to a normal arrangement, and considerable degeneration of nerve fibers and ganglion cells throughout the cortex. He believes that the nerve elements are primarily affected, and that the histological changes are sufficient to explain the dementia. Other cases of so-called genuine epilepsy exist without these characteristic changes, but the cases such as he describes, which he calls the Chaslin form of epilepsy, are not rare. It is probable that different diseases are included under what we now call epilepsy. The illustrations accompanying this paper are exceedingly interesting.

SPILLER.

71. RECHERCHES SUR LES LÉSIONS DES CENTRES NERVEUX, CONSÉCUTIVES À L'HYPERTHERMIE EXPÉRIMENTALE ET À LA FIÈVRE (Studies of the Effects of Fever and Artificial Hyperthermia on the Nervous System). G. Marinesco (Revue neurologique, 7, 1899, p. 3).

Goldscheider and Flatau, Brasch, Moxter, Ewing and others have shown that, both in diseases attended with high temperatures, and in artificial raising of the temperature in animals, certain changes, similar in character, are present in the ganglion cells of the nervous system. In this present contribution, Marinesco comes to the conclusions that there are at least three stages in the development of complete chromatolysis, some of which he thinks have escaped other

observers. In the first stage, the perinuclear elements and the nucleus show no marked variations; the prolongations, however, are not colored. These alterations are reparable. They are caused by high temperatures acting for a short time only. The second and third stages have been thoroughly reported upon by the observers above mentioned; but Marinesco thinks that these observers, for the most part, have not differentiated with sufficient care between lesions which result from high temperatures and lesions which might be produced by toxic agents in patients with high temperatures, as in pneumonia, typhoid and other infectious diseases. Thus in such cases, are the lesions found due solely to the increased temperature or to the toxic agent alone, or both? Marinesco believes that we are not yet in a position to decide definitely upon these questions. His general conclusions drawn from the present series of observations may be expressed as: (1) Temperatures below 40° C. (104° F.) in man, even if continued for several days, do not produce lesions as definite as in experimental hyperthermia in animals. (2) In febrile infectious cases, lesions may be found which do not belong to the temperature changes alone, since in experimental work in animals, similar changes do not seem to occur. (3) In cases in which the temperature has reached 41° C. (105.8° F.), and this has been maintained for several hours, lesions are found similar to those in experimental hyperthermia.

JELLIFFE.

72. UNILATERAL CHANGES IN CEREBRAL HEMORRHAGE, EMBOLISM AND THROMBOSIS. Williamson (*British Medical Journal*, 1, 1898, p. 1515).

In addition to the fundus changes currently conceded to be present with more or less rarity in sudden vascular lesions of the brain (albuminuric retinitis, embolism of central artery, narrowing of arteries, etc.), the author found retinal hemorrhages or dilatation of the retinal vessels on the side of the lesion in eight of thirteen cases of fatal cerebral hemorrhage or vascular occlusion, while the fundus on the opposite side was normal. In three cases both fundi were normal and in two albuminuric retinitis was present.

PATRICK

CLINICAL NEUROLOGY.

73. ON BERI-BERI OCCURRING IN TEMPERATE CLIMATES. Conolly Norman (*British Medical Journal*, Vol. 11, 1898).

After showing that the disease is not limited to the tropics and that in temperate climates it is particularly apt to occur in asylums, the author proceeds to describe the disease as observed by him at the Richmond Asylum, Dublin.

The first symptom observed was usually edema over the tibia, and generally cardiac disturbance was present from the very first. Dealing with a large number of cases, the diagnosis was roughly based on the following combination:

(1) Edema of the shins, without albuminuria; (2) tachycardia and cardiac irregularity; (3) pains in the legs; (4) anesthesia of the integuments of the legs. Of these it was somewhat difficult, owing to the mental condition of the patients, to estimate the degree of pain, etc., and often impossible to be sure of the existence of anesthesia.

Very frequently there was marked cardiac irritability and weakness and respiration was often thoracic, apparently from feeble action of the diaphragm.

Of the motor symptoms the earliest and most marked was paresis

of the anterior tibial and peroneal muscles, causing foot-drop and *steppage*. In addition to this, weakness of arm and thigh muscles was frequent: wrist-drop occurred in less than one per cent. of the cases. Unilateral paralysis of the third nerve was observed once.

Sensory symptoms were those ordinarily found in multiple neuritis, painful formication being the most frequent, but the author mentions in addition, what he has not seen previously described, hypæsthetic zones surrounded by an area of distinct hyperæsthesia. The reflexes, both superficial and deep, were generally exaggerated in cases that came under observation early. As the case progressed the knee-jerks usually diminished and when there was marked loss of power they were always abolished.

Course of the Cases.—Usually the cases began insidiously. In the sane a feeling of weariness in the legs, with occasional cramps, often preceded more definite symptoms for a considerable time. It was generally difficult to obtain evidence of prodromal symptoms among the insane. Sometimes the earlier symptoms were accompanied by a smart rise of temperature, but this soon subsided. Two cases began during convalescence from typhoid, and several patients were attacked after recovery from dysentery. The course of the affection was extremely variable in different cases. Sometimes it was steady, much more often jerky. Quite characteristic were the features observed in other epidemics of beri-beri: sudden changes for the worse in cases which were apparently doing well; extreme frequency of relapse; occasional quite sudden deaths, not infrequently occurring when convalescence had been apparently progressing favorably. The length of an attack was perfectly uncertain; on the whole the severer cases of the first epidemic tended to terminate, either favorably or the reverse, more quickly than the milder cases of the later epidemics. Speaking generally, the cases tended in the early stage to increasing dropsy and increasing loss of power, then the dropsy passed off and marked muscular wasting was apparent, which again gradually became rectified, paralysis and anesthesia passing off at the same time. Death occurred in a variety of ways. That terrible condition, so often described in connection with beri-beri, known to the Japanese as “*shiyo-shin*,” and apparently dependent upon failure of an overworked and weakened heart, carried off several patients, after longer or shorter periods, often coming on with fulminating rapidity, of extreme suffering (restlessness, vomiting, violent pulsation of heart, dyspnea, cyanosis, orthopnea, etc.). Death was often due apparently chiefly to edema of the lungs. Syncope, apparently due to degeneration of heart muscle (found after death) was not a rare cause of death. Sometimes, as mentioned, this mode of death occurred in a case which had been apparently convalescing. Hydropericardium and hydropleura probably contributed to an unfavorable termination in several cases. In some cases more general exhaustion seemed to be the immediate cause of death. This occurred particularly among the old and debilitated. Sufferers from beri-beri readily succumbed to other diseases. Paralysis of the respiratory muscles, particularly the diaphragm, alone or in combination with heart failure, sometimes caused death.

Edema was, the author believes, a constant condition, though in a few cases it was very slight at first or even escaped attention, and the patient appeared to pass directly into the “dry” or atrophic state. In slight cases and in early cases it was most marked along the inner side of the tibia, forming the pear-shaped swelling of *Pekelharing*. Less constantly the feet and legs generally were swollen. Edema could usually be detected also over the sacrum, often over the sternum or ribs, over the ulnar border of the forearm, and not infrequently, even in slight cases, the face was puffy. Some cases presented gen-

eral anasarca, often extreme. Edema of the lungs was common, and hydropericardium and hydropleura also occurred. The superficial edema was curiously variable: in some cases pitting was readily produced, in others there was doughy, myxedematoid condition with much swelling and little pitting. In marked cases the edema was sometimes noted to shift with posture more readily than is common in other forms of anasarca. It also disappeared in some cases with singular rapidity. Edema of the fundus oculi was found in some cases and occasionally effusion into the knee joint was noted. PATRICK.

74. "REMARKS ON A CASE OF PORENCEPHALY." Gibson and Turner, (Edinburgh Medical Journal, 2, 1898, p. 164).

The authors give the history and post-mortem findings, in the case of an imbecile woman of twenty-two, who, when three days old, had been seized by convulsions and had since been epileptic. She was admitted to the hospital in status epilepticus, a peculiarity of the recurring convulsions being that they were confined to one side of the body, and alternated, so that there would be two convulsions affecting the left side, followed by one affecting the right side. Death occurred about eight hours after admission. The necropsy showed a defect of the right half of the cerebrum. The occipital, and part of the parietal, and temporo-sphenoidal lobes were cystic, the gyri over this area being atrophied, and the convolutions of the remainder of this side of the brain presenting a curiously shriveled appearance. The *left* cerebellar hemisphere was also atrophied. This is in harmony with a crossed cerebro-cerebellar connection previously described. The article is illustrated by three plates. ALLEN.

75. SELTENE ERSCHINUNGEN AUF OPTISCHEM GEBIET IN GEFOLGE VON APOPLECTISCHEN INSULTEN (Extraordinary Optical Symptoms as a Result of Apoplectic Injuries). Richard Hilbert (Memorabilien, 1898, p. 321).

A scientific man, a botanist of the highest education, and a most experienced observer, aged sixty-nine, had suffered in his sixty-third year with a very slight attack of apoplexy, and afterwards with several attacks of about the same degree. At the beginning of the present year he had another slight attack, apparently not more than a passing loss of consciousness, following which very striking psychical symptoms appeared. At the time of the examination there was a contraction of the right arm and hand, the right leg was weak, the pulse tremulous, tongue deviated to the right, the pupils were alike, moderately dilated, re-acting to light, both sight and hearing somewhat impaired. The lens of the eye was perfect, the eye ground showing nothing but senile alterations. Slight aphasia, the patient mixing words or using the wrong word. He was likewise agraphic, and understood matter read with difficulty or not at all. His subjective symptoms were the interesting features of the case. On waking in the morning he saw the whole of his room blue. He declared that the hands of his visitors were misshapen and enlarged. He could not understand why no one would agree with his observations. He sometimes said that other small objects were enlarged or diminished. He was most disturbed by certain hallucinations of smell or hearing and especially of sight. He complained constantly that some one had been smoking in his room, a practice to which he had particular objection. Again, he noticed almost constantly appearing strange shapes

of tiny beasts. These sometimes staid still in one place and sometimes ran with inconceivable swiftness and hissing noises about his room from one corner to the other. If these disappeared after a time, almost immediately there appeared in their places naked women, and he thought it strange that the ladies of his family should regard them with any calmness. His memory for recent events had almost entirely disappeared, and his previously unusual intellect was brought down to a very low level. Being informed of the entirely subjective nature of his visions he began to observe them with interest, but still with a certain doubt if they were not, after all, real. Thus, besides the ordinary symptoms of a cerebral apoplexy in the optical territory, and apart from the frequent hallucinations, he had three unusual symptoms, cyanopia, macropsia, and metamorphopsia. All of these are so rare that it is the more extraordinary that they should all have occurred in the same patient. The variety of the symptoms is also remarkable: Paralysis, contracture, tremor, aphasia, agraphia, lessening of sight and hearing, intellectual decay, partial loss of memory, hallucinations of sight, hearing, and smell, and finally, besides all these, cyanopia, macropsia, and metamorphopsia.

MITCHELL.

76. AN ACUTE CASE OF LANDRY'S PARALYSIS. Henry H. Haward (*British Med. Jour.*, I, 1898, p. 1654).

A man of fifty-four years had been for a week exposed to the weather more than usual. After having felt a tingling in the hands for two hours, he walked a mile to his home, felt disinclined for food, went to bed, could not sleep and three hours later found that food would not go down. After this he rapidly lost strength so that in twelve hours he was quite helpless, respiration was hurried and stridulous and both deglutition and expectoration were impossible. Deep and superficial reflexes were present and although he had no pain, tingling and numbness were very distressing. Anesthesia was absent throughout. The muscles were affected symmetrically, and approximately in the following order: First, the pharyngeal, laryngeal, spinal and cervical; then those of the hips, thighs, shoulders and arms; next, the lower intercostal and abdominal; followed by those of forearms, legs, hands, feet, and upper ribs; lastly, facial, lingual, labial, and diaphragmatic muscles. The patient died forty-three hours after the appearance of the first symptom.

PATRICK.

77. APHASIA IN RELATION TO TESTAMENTARY CAPACITY. (*British Medical Journal*, September 3d, 1898).

In a discussion on this subject at the last meeting of the British Medical Association, Gairdner said that no general or positive rules could be laid down. It was not a general question of law; not a thing that could be put into legal or physiological categories. It was a question of detail in each individual case and, we gather, to be decided pretty much without direct reference to the aphasia. Does the testator know what he wants? Does he form a clear conception of what he wants and does he succeed in giving effect to that conception in the particular document that is the "will?" Or is the document not really his will at all, but unduly inspired by others? These are the questions of detail to be decided.

William Elder in a much more systematic consideration of the

subject comes to the following rather categorical, somewhat theoretical, but, on the whole, apparently safe conclusions:

1. That organic diseases of the brain may render a patient incapable of making a will, and that some form of aphasia may be produced also as one of the symptoms of the organic disease.

2. That some forms of aphasia may render a patient incapable of will making.

3. That auditory aphasia, if at all well marked, incapacitates a patient from will making.

4. That some other forms of aphasia, such as pictorial word-blindness, pictorial motor aphasia, and graphic aphasia, may render a patient incapable of making a will, not necessarily from being mentally incapable, but from the difficulty of carrying out the legal formalities.

5. That these difficulties in carrying out the legal formalities necessarily vary according to the law of the particular country.

6. That simple uncomplicated cases of infrapictorial auditory, infrapictorial visual and infrapictorial motor aphasia are capable of valid will making.

Clouston insisted upon two points.

The first was the test question, whether it was the will of the individual, or whether it had been suggested to him. The second was that in making the will of any aphasic patient it was the duty of every medical man and lawyer to put the contrary case. A man had left, say, £100 to his wife and £100 to his daughter—to A. and B. They were bound to ask him if it was for B. and C., or for D. and E., that he intended the money. No will of an aphasic, in spite of any judge or doctor, could be a legal and proper will unless the doctor had put the contrary case, because an aphasic would assent to anything if put to him in a certain way. It was essential that the mental condition of the would-be testator should be tested by a medical man. Dr. Clouston concluded by urging that two practical points in the actual making of the will must be attended to: (1) Test whether the document is his own, and has not been suggested by others; (2) Put the contrary proposition, or a different proposition, twice at least as to sums disposed of, and as to the persons to whom the money or property is left. No will can be valid where these two points have not been most carefully gone into.

PATRICK.

78. *CONTRIBUTO ALLA CONOSCENZA DELLA PROCESSOMANIA, STORIA DI UNA DEGENERATA* (Contributions to the Study of Litigation Mania). Sante de Sanctis (Revista sperimentale di Freniatria, 24, 1898, p. 350).

The author has been enabled to follow the history of this family since 1754. Throughout the whole family there has been marked mental insufficiency, with exaggerated ideas of property and most of the members in the several branches of the family have indulged in law suits on various small pretexts.

JELLIFFE.

79. *UN CAS PARTICULIER DE CÉCITÉ PSYCHIQUE* (A Peculiar Case of Mind-Blindness). Pauly (Lyon Médical, 88, 1898, p. 364).

A case of what Kussmaul called apraxia; Hughlings Jackson, imperception; and Wernicke, asymbolism; that is, the patient was as a rule unable to recognize objects by sight, although generally recognizing them at once by the sense of touch. The condition followed a

series of slight epileptic attacks without hemiplegia. The patient had a right hemianopsia, some impairment of memory and difficulty in awaking visual images of objects, but no aphasia, strictly speaking. PATRICK.

80. TEMPERAMENTO E CARATTERE NELLE INDAGINI PSYCHIATRICHE E D'ANTROPOLOGIA CRIMINALE (Character and Temperament in the Insane and in Criminals). Del Greco (Il Manicomio moderno, 14, 1898, p. 161).

The author makes two general categories for human kind; the one, psycho-physiques, or temperament, and psycho-social, or character. A man's inner feelings determine his temperament; his relations to his fellow men, his character. The author holds, that from both points of view, in many neuroses there are well established variations in both temperament and character, which are closely correlated to the neurosis in question; thus he would speak of an epileptic or paranoiac temperament and character. In the criminal there are changes of much the same type, so that while a criminal may not be epileptic nor be a paranoiac, yet he usually possesses a temperament and a character which approaches those of the epileptic and the paranoiac. JELLIFFE.

THERAPY.

81. A DISCUSSION ON THE THERAPEUTIC VALUE OF RECENT SYNTHETIC ANALGESICS; THEIR BENEFITS AND ATTENDANT RISKS (British Medical Journal, No. 1971, 1898, p. 1054).

Ralph Stockman thought that none of the analgesics hitherto tried acts in an ideal way or is without serious drawbacks for general and frequent use. He prefers phenacetin as being less harmful than acetanilid or even than antipyrin. Lactophenin is almost identical with phenacetin.

C. D. F. Phillips: Analgene, although it cannot be considered a very powerful analgesic agent, yet has the advantages of but slight toxicity, of tastelessness, and of easy solubility in acid media, so that, being readily absorbed from the stomach, its action is quickly produced. On account of its tastelessness and comparative harmlessness it is of especial value in the treatment of painful conditions in children. Its effect is transient, but this, if it be a defect, is easily overcome by a frequent repetition of the dose. Its great evil depends on the destructive action it exerts on the red blood corpuscles resulting in urobilinuria. Although untoward effects such as nausea, vomiting, diarrhea, tremor, vertigo and rashes have followed its use, yet, as a matter of experience, these unpleasant accidents rarely occur.

Euphorin is without doubt a powerful analgesic; it is said to have the activity of twice its weight of antipyrin. But like some other powerful analgesics it tends to interfere with the respiratory processes and to weaken the heart's action, and produce cyanosis with collapse. A negative advantage it has is that it does not induce changes in the blood or affect the kidneys.

Methylene blue is a distinctly useful analgesic, particularly in functional neuralgias and in all kinds of nervous headache. It, however, changes the hemoglobin of the blood into methemoglobin, and causes irritation of the stomach, leading to vomiting and diarrhea, and also of the urinary tract, having caused in different cases albuminuria, retention of urine, stranguary, cystitis, and spasmodic con-

traction of the bladder. In large doses it has caused muscular paresis, loss of sensibility and dyspnea.

Agathin is said to be both slow in its action and unreliable in its effect. It has caused vomiting, diarrhea, insomnia, vertigo, loss of consciousness, and even increase of the pain which it was given to relieve. It is probably not worthy of a place among valued analgesics both on account of its limited applicability and because of the accidents which have followed its employment.

The compounds of antipyrin, namely, pyramidon, salipyrin, toli-pyrin, tolisall and ferripyrine, have, as analgesics, merely a weak action of their antipyrin element. They are harmless, but not as a rule sufficiently powerful for the treatment of urgent pain.

Those which are derived from salicylic acid, as well as antipyrin, namely, salipyrin and tolisall, are of use in painful states of rheumatic origin, but otherwise they are comparatively unimportant.

Malakin, being only slightly soluble in the ordinary menstrua, is slow in its action, very much slower than phenacetin, antifebrin or antipyrin, and is in every way much inferior to them; with the exception, however, that it may be given for a long period of time without disordering digestion. It has been found of distinct service in acute rheumatism. It has no deleterious action on the blood or kidneys, being indeed practically free from risk.

Neurodin, one of the para-amido-phenyl derivatives of anilin, is another remedy which is feeble in its action, but harmless in doses sufficiently large to elicit its analgesic effect. Not only is it weak, however, but it is uncertain, and must be considered greatly inferior to phenacetin and antipyrin.

Lactophenin appears to be more active as a mere sedative to the nervous system (relieving irritability, restlessness and depression) than as a pure analgesic. It is comparatively free from risk, although in medicinal doses it has produced vomiting, coldness of the body surface, cyanosis and collapse. But its great disadvantage is that it is inconstant in its analgesic effect; it has been known to fail completely in relieving the pain of multiple neuritis and of intercostal neuralgia.

Citrophen has not so far been much used as an analgesic. Its applications are much the same as those of phenacetin, but its attendant risks are greater, while its benefits are much less distinct. It has exerted in medicinal doses a gravely toxic effect on the blood, and has produced serious irritation of the intestine and kidney. It probably ranks very much below phenacetin.

Apolysin closely resembles phenacetin in its effects; it is said to produce analgesia even more vigorously than that remedy, but to be likewise more toxic. As it is much more soluble than phenacetin and the other members of the same group its action is correspondingly more prompt. It has been of especial benefit in migraine in diminishing the violence of the pain, in allaying the cutaneous hyperesthesia, and in shortening the duration of the attack. It is well tolerated in large doses, continued over long periods of time, without showing any tendency to accumulation or produce intestinal or renal irritation, or indeed any material injurious result. Its toxic effects, which are those of phenacetin, but more pronounced, are especially liable to occur if it is given on an empty stomach, or when that organ is producing an excessive amount of acid secretion. It is a remedy which, I think, may prove a valuable supplement to the phenacetin-antipyrin-exalgin group.

Phenocoll hydrochloride is a derivative of phenacetin, and is said to be as powerful an analgesic as it, and, being more soluble, it acts more quickly. Even if this be granted, it cannot be considered

so safe a remedial agent. Its special use has been in neuralgias brought on by cold, that is, of rheumatic origin. But in children, and in debilitated adults, more especially in cases of advanced phthisis, it has caused alarming collapse with cyanosis. In other cases it has produced labial herpes, vertigo and general malaise. It is, however, a substance which, and as I expect, may take an important place in the future as a means of relieving painful states generally.

The salicylate of phenocoll or salocoll has no advantage over the hydrochloride of phenocoll except that advantage which is due to the action of its salicylic acid element. It is said not to cause any gastric irritation, or indeed any untoward effect when given in medicinal doses.

Salophen is probably the best of the recent synthetic analgesics. It was introduced to replace salol, and has instead of its phenol radicle the acetyl-para-amido-phenyl radicle. Like salol it is not decomposed until it reaches the intestine, so it does not produce any gastric disturbance, as do the salicylates. Its toxicity is very slight, and is apparently due, when given in very large doses, to the salicylic acid it contains, but in medicinal doses it does not even produce the tinnitus and headache characteristic of the action of the salicylates. It has been of benefit in painful states of all sorts, but of special value in the neuralgias of children. When it is used to relieve the pains of influenza it is necessary to continue its administrations for a few days after the pains have been relieved. It is held, and with considerable reason, to be as effective and as safe as, or safer than, antipyrin and phenacetin, and it is probable that before long it will take rank with these substances.

Conclusions.—I would submit that salophen, phenocoll hydrochloride, apolysin and methylene blue are substances of high therapeutic value, and, without attempting to arrange them in order, would single out salophen as of exceptional promise; that agathin, being slow, unreliable, and even dangerous in its action, should be avoided; that some hesitation should be exercised in employing analgene and citrophen because of their toxic action on the blood, in using euphorin and lactophenin because of their inconstancy and tendency to produce collapse, and in choosing malakin because of its tardy action; and that pyoktanin, ferripyrin, pyramidon, salipyrin, tolipyrin, tolisall, and even salocoll, although they may be well and safely used as substitutes for better remedies, are unnecessary.

A. Lockhart Gillespie called attention to the benefit of adding caffeine to the analgesics and recommended migrainin as a good analgesic with no deleterious action on the heart.

D. J. Leech had had very good results from antipyrin and phenacetin. He considered pyridin decidedly harmful and had seen evil results follow the use of exalgin.

John Liddell had used exalgin extensively and had seen no ill-effects when given in proper doses.

J. R. Hamilton never used phenacetin or antipyrin without caffeine.

William Gordon had prescribed antifebrin for many years with no harmful results and also commended exalgin and phenacetin.

W. S. Frew used three or four of the chief drugs indifferently and considered antifebrin no more dangerous than antipyrin except perhaps because of its smaller dose.

J. O. Affleck thought exalgin of great service in reducing pain, but had seen fewer ill-effects produced by phenacetin than by any other of these drugs.

In closing the discussion, Dr. Stockman pointed out the danger of giving such substances as phenylhydrazin and anilin, or their de-

rivatives, to a patient *de novo*. He was inclined to agree with Dr. Frew in thinking that as caffeine produced high tension and stimulated the cerebrum and cord it was rather to be avoided. He pointed out that the depressant action of analgesics was not, as some of the speakers had seemed to indicate, directly due to their action upon the heart, but to their influence upon the vasomotor center, and it was this action which was affected by caffeine. The little known drugs had their uses, and they should be thoroughly investigated before being abandoned. He said that Dr. Liddell's desire for a local anesthetic would probably never be fully satisfied; in the meanwhile, ethyl chloride and cocaine were all that were required. PATRICK.

82. THE TREATMENT OF HYDROCEPHALUS BY INTRACRANIAL DRAINAGE. G. A. Sutherland and W. Watson Cheyne (Brit. Med. Jour., No. 1972, 1898, p. 1155).

The authors concluded from the investigations of Leonard Hill that in cases of hydrocephalus if an outlet were provided for the fluid contained in the ventricle so that it might flow into the meningeal spaces, it would rapidly be absorbed by the veins until the cerebral venous pressure and the cerebro-spinal pressure were equalized. In pursuance of this theory they operated upon three cases of congenital hydrocephalus, draining from the ventricle into the sub-dural space by means of catgut.

In the first case the diminution in the size of the head was rather rapid and continuous, but there was no improvement in the infant's mental or physical development, and it died three months after the operation with symptoms of basilar meningitis.

At the necropsy a considerable quantity of straw-colored fluid was found in the sub-dural space. The ventricles were both dilated, but not distended with fluid. The membranes at the base were thickened, and the ependyma of the ventricles formed a distinct membrane of a dark purple color, firm but not granular. The opening into the left ventricle made at the operation was quite evident, and some strands of catgut lay in it, while the rest of the drain could be traced up to the opening made in the dura mater, at which point the brain was adherent to the dura mater. The adhesions formed were soft and easily broken down, and amongst them lay the external ends of the catgut drain. The brain was soft, cystic in parts, and very imperfectly developed.

In the second case there was also prompt and rapid diminution in the size of the head until all the bones of the cranial vault were overriding. At the end of four months the head was noticed to be quite asymmetrical and upon shaving the hair it was discovered that the right side of the fontanelle was tense and fluctuating while the left half gave no evidence of tension. It was apparent that drainage of the left ventricle was complete while that of the right had come to a standstill. Accordingly an operation was performed on the right side similar to that which had been done on the left and this operation was followed by complete disappearance of intracranial tension and gradual diminution in the size of the right side of the head. Six months after the first operation there was overriding of the cranial bones and the child had improved physically, but there was no evidence of mental development.

In the third case there were no apparent results from the operation and it was repeated a fortnight later, also without apparent results. Two weeks after this second operation the child died of measles.

In the discussion, Doctor Stiles reported three operations, all of which terminated unfavorably. PATRICK.

Book Reviews.

TRAITÉ PRATIQUE DE RADIOGRAPHIE ET DE RADIOSCOPIE, TECHNIQUE ET APPLICATIONS MÉDICALES. A. Londe, Director of the Photographic and Radiographic Department of the Salpêtrière Hospital; Laureate of the Academy of Medicine; of the Faculty of Medicine of Paris. Gauthier Villars, Paris, 1898, pp. 244.

The well known reputation of this author as an expert photographer of medical cases makes him particularly well fitted to give information regarding this new and important method of diagnosis. His book is a most complete and practical exposition of the technique and application of Roentgen's discovery to medicine and surgery. While dealing very practically with each step, it is not so long as to be tedious or so short as to be valueless, each part receiving its full share and consideration.

The illustrations of apparatus are profuse and well explained, and give the reader a knowledge of their uses, varieties, and the conditions which make the employment of each necessary and most valuable. We note, however, that the self-regulating shunt circuit tube which was devised and perfected in this country is reproduced with some slight modifications, but without giving credit to its inventor, Mr. Sayen. Its use is not appreciated either, for the self-regulating device is only described as adjusting it to the particular coil or apparatus used to energize it. Its value in producing the different qualities of Roentgen discharge seems to be entirely unappreciated, and as we look further through the book there is no mention made of the different qualities of the Roentgen discharge produced by varying vacua, and of the varying results that may be produced by their employment. This lack of knowledge probably accounts for the statement later on that the diagnosis of calculous nephritis is impossible. The absolute positive and negative diagnosis of this condition is possible and depends on the varying qualities of the Roentgen discharge, a low vacuum discharge being required.

We note, however, with pleasure that the author insists that the localization of foreign bodies is practical, and that mathematically accurate results can be attained if precise methods and instruments are employed. This chapter is very instructive, and the methods employed have a simple, yet absolute foundation on mathematical principles.

In the application of the Roentgen ray method of diagnosis to medicine and surgery, we note its employment in determining the lack or arrest of development of the osseous system in certain affections. The author illustrates this by a case of myxedema which shows in a series of skiagraphs the changes produced by thyroid treatment. The study of the different forms of infantilism is also practical in its value. Anomalous developments in the osseous system are noted. Observations have been made in cases of macrodactylitis and infantile paralysis; in hemiplegia and atrophy the bones of the sound and affected limbs have been compared and the differences even of their structure distinguished. As an illustration, a case of de-

forming osteitis of Paget is beautifully depicted. The condensing and rarifying varieties can also be separated.

The deformities of gouty and rheumatic arthritides can be distinctly perceived and differences noted. In the application to the thorax we find an omission of thoracic aneurisms and their differentiation from new growths of the mediastinum.

The various surgical applications have their due consideration, and the value of this method of diagnosis receives in this book a substantial confirmation.

The student will find it a work that is of great value to him in gaining the requisite knowledge of the technique of this method of diagnosis.

CHARLES LESTER LEONARD.

VERMISCHTE AUFSÄTZE. (V. Heft der Neurologischen Beiträge), von P. J. Möbius, Leipzig. Johann Ambrosius Barth, 1898, pp. 173.

As indicated in the title, this is the fifth volume of neurological fragments that have come from the author's pen. As stated in the preface, the others have had their readers, if not many, still appreciative ones, and the writer hopes that this new volume may have as fortunate a fate. That it will, we can have little doubt, both from the suggestiveness of much that it contains and from the variety of its subject-matter.

Some of the topics discussed are: The Classification of Diseases; Hemihypertrophy; The Treatment of Persons with Nervous Disease, and the Arrangement of Institutions; Short Sketch of a Monastery; Remarks Concerning Priority; The Causes of Diseases; Concerning Smoking; In Memoriam, J. M. Charcot; and many other sketches of varied character.

In all this variety there is something of interest to everyone. Facts of daily life and of medical import are regarded from the point of view of the philosopher, and as philosophical reflections the author no doubt intends his essays to be judged. Considering the necessity that most of us are under to spend our time and mental effort in the grasping of multitudinous facts, it is a relief, at times, to read something in the general field of medicine, of more literary pretensions. Möbius' book affords a certain satisfaction to this want, and to those who wish to vary their professional reading with the semi-scientific essay, we do not hesitate cordially to recommend this collection.

E. W. TAYLOR.

JAHRESBERICHT ÜBER DIE LEISTUNGEN UND FORTSCHRITTE AUF DEM GEBIETE DER NEUROLOGIE UND PSYCHIATRIE. Herausgegeben von Dr. E. Flatau und Dr. L. Jacobsohn in Berlin, Redigiert von Professor Dr. E. Mendel in Berlin. Erster Jahrgang. Bericht über das Jahr 1897. Verlag von S. Karger, Berlin, 1898. 30 marks.

There is probably no domain of the medical sciences that needs a more painstaking and thorough research than that of neurology, and of late years the workers in this chosen field have been more than usually active. Thus a positive necessity has arisen for some sort of a general reference work of wider scope and better classification than was afforded by the standard journals in the French, German, Italian and English languages. When it is borne in mind that there are at least 60 journals alone devoted to the study of neurology and psychiatry and its related branches, it is evident that a "Jahresbericht" has become a crying need. S. Karger, of Berlin, has furnished us with this volume, and medical science in general, and neurological

science in particular, will be greatly benefited. The work is ably edited by workers whose zeal and knowledge have been commensurate with the prodigious task, and the results are more than gratifying in a volume of 1,600 pages.

The arrangement and classification of titles and abstracts is about as follows: About 1,100 pages are devoted to Neurology, although the lines are not drawn overstrict, and the remaining 500 to Psychiatry. The general microscopical technic of the examination of the nervous system is first taken up. Then follow, Anatomy of the Nervous System; Physiology, both general and special; Pathological Anatomy of the Nerve Cells and Fibers of the Peripheral and the Central Nervous System; the general pathology of the nervous system is treated of under the following heads: General Symptomatology, Etiology, Diagnosis, etc.; Diseases of the Spinal Cord; Diseases of the Cerebrum and Cerebellum, the Pons and the Medulla; Diseases of the Peripheral Nerves, and Functional Diseases. Part 6 deals with the Therapy of the Nervous System. Under the general heading of Psychiatry are found: Psychology, General Etiology, Symptomatology, and Diagnosis of Mental Disease, special portion, which treats the several types of mental diseases consecutively; Criminal Anthropology, Forensic Medicine, and finally, the Therapeutics of Mental Diseases. The general works in neurology and psychiatry that have appeared throughout the year 1897 are reviewed at the close of the book, and a subject and name register close the volume.

We have for the undertaking nothing but unqualified praise both for the publishers and the collaborators. One single blemish that appears to the present writer is the absence of much work appearing from good workers, but which is published in obscure journals and in languages which are usually inaccessible to the majority of mankind. Most workers in neurology and psychiatry to whom this work would be of any value at all are fairly conversant with French, German and English, and articles appearing in these languages are easily obtained and referred to; whereas much of the Russian, Italian, Swedish, Hungarian, Greek and other work is lost absolutely unless presented by such a *Jahresbericht*. And we would suggest that greater attention be paid to the work which is harder to get at by the special worker and that the more readily accessible material be referred to by name only or by short abstract. It would appear ungracious to ask for any more than is presented in this volume, yet we believe that there is some value in the suggestion here brought forward.

JELLIFFE.

THE PRINCIPLES AND PRACTICE OF MEDICINE, DESIGNED FOR THE USE OF PRACTITIONERS AND STUDENTS OF MEDICINE. By William Osler, M.D., Fellow of the Royal Society; Fellow of the Royal College of Physicians, London; Professor of Medicine in the Johns Hopkins University and Physician in Chief to the Johns Hopkins Hospital, Baltimore; formerly Professor of the Institute of Medicine, McGill University, Montreal; and Professor of Clinical Medicine in the University of Pennsylvania, Philadelphia. Third edition, entirely revised and enlarged. New York: D. Appleton & Co.

The great popularity and wide-spread use of this most admirable treatise makes an extended discussion of its excellencies a work of supererogation. The present edition has been much enlarged and a great deal of valuable matter added. The following articles have been rewritten or are new: Vaccination, Beri-Beri, the Bubonic Plague, Cerebro-spinal Fever, Pneumonia, Malta Fever, Yellow Fever, Dengue, Influenza, Leprosy, Glandular Fever, the Gonorrhœal Infection, Cancer

of the Stomach, the Gastric Neuroses, Enteroptosis, the Cirrhoses of the Liver, Jaundice, the Diseases of the Bile Passages, the Diseases of the Pancreas, Diseases of the Thymus Gland, Diseases of the Spleen, Lymphatism, Addison's Disease, Encephalitis, Neurasthenia, Erythromelalgia and many shorter articles. Changes in arrangement have also been made, notably in the section on nervous disease.

The systematic arrangement and thoughtful treatment of the subject matter greatly facilitate the labors of the student, while the writers' delightfully lucid and interesting style makes the book one to be read with pleasure by the practitioner as well. Although Prof. Osler's views on some subjects differ from those of other writers, the work is thoroughly modern and accurately represents the present state of medical knowledge. The mechanical make up of the book is excellent, and type and paper leave nothing to be desired.

JELLIFFE.

BOOKS RECEIVED.

"Second Annual Report of the Loomis Sanitarium for Consumptives," Liberty, N. Y.

"Report of the Trustees of the Mass. Hospital for Epileptics," Palmer, Mass.

"Twenty-Sixth Annual Report of the State Charities Aid Ass'n," New York.

"Fifth Biennial Report of the Delaware State Hospital," Farnhurst, Del.

"Sixth Annual Report of the Sheppard Asylum," Baltimore, Md.

"Twelfth Biennial Report of the State Lunatic Asylum No. 2," St. Joseph, Mo.

"One Hundred and First Annual Report of the Maryland Hospital for the Insane," Catonsville, Md.

"Third Annual Report of the Second Hospital for the Insane of the State of Maryland."

"Chandler's Encyclopedia," in Three Volumes, by Wm. H. Chandler, Ph.D. Peter Collier, N. Y.

"Diseases of the Eye," by G. E. deSchweinitz, M.D. W. B. Saunders, Philadelphia, Pa.

"Guy's Hospital Reports," Vol. LIII. J. & A. Churchill, London, Eng.

"An American Text-Book of Diseases of the Eye, Ear, Nose and Throat," by G. E. deSchweinitz, M.D., and B. Alex. Randall, M.D. W. B. Saunders, Philadelphia, Pa.

"A Text-Book of Mechano-Therapy, Massage and Medical Gymnastics," by Axel V. Grafstrom, M.D. W. B. Saunders, Phila., Pa.

"Sajous' Annual and Analytical Cyclopedic of Practical Medicine," Vol. 2. The F. A. Davis Co.

"The American Year Book of Medicine and Surgery," by Geo. M. Gould, M.D. W. B. Saunders, Philadelphia, Pa.

"The Dawn of Reason," by James Weir, Jr., M.D. The Macmillan Co., N. Y.

"Suggestion und ihre sociale Bedeutung," by W. von Bechterew. Arthur Georgi, Leipzig.

"Die Leitungsbahnen im Gehirn und Rückenmark," by W. von Bechterew. Arthur Georgi, Leipzig.

"Neurologie des Auges," by Dr. H. Wilbrand and Dr. A. Saenger. J. F. Bergmann, Wiesbaden, Germany.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

RETROBULBAR NEURITIS AND FACIAL PALSY OCCUR-
RING IN THE SAME PATIENT, WITH CASES.¹

By G. E. DE SCHWEINITZ, M.D.,

PROFESSOR OF OPHTHALMOLOGY IN THE JEFFERSON MEDICAL COLLEGE,
PHILADELPHIA, PA.

As is well known an interstitial optic neuritis, most severe in the optical canal and at first chiefly located in the papillo-macular tract, from which it may extend, however, through the whole diameter of the nerve, usually manifests itself by the following symptoms: Obscuration of vision, beginning in the center of the visual field and rapidly progressing to complete or nearly complete blindness; either negative ophthalmoscopic appearances, or at most blurring of the margins of the disc, hyperemia of its surface and alterations in the caliber of the retinal vessels, the arteries being diminished in size and the veins distended and pulsating; and marked pain on movement of the eyeball, or when the globe is pressed backward into the orbit. Such an affection may arise because of the presence in the blood of an infecting agent existing in association with some disease, for example, rheumatism, gout, syphilis, influenza, scarlet fever, etc., or because this infecting agent comes directly from a diseased focus in the mucous membrane of the nose, the ethmoidal cells or the sphenoidal sinus, or arises as the

¹Read before the Philadelphia Neurological Society, Feb. 27, 1899.

direct result of an inflammatory process in the orbit, *e.g.*, cellulitis, or in the optical canal, *e.g.*, periostitis, gummatous deposits, etc. The same disease at times appears to be the result of certain toxic agents, such as alcohol or lead, or may be provoked by menstrual disturbances, especially sudden suppression of the menses, by "catching cold," or by overwork and prolonged eye-strain. That a similar ocular disease is at times part of the symptomatology of an insular sclerosis, or of an acute or subacute myelitis, and is then of most serious prognostic import, need not be more than mentioned in this society.

Retrobulbar neuritis may be acute, subacute or chronic, and the acute cases are at times exceedingly rapid in onset and development, forming the fulminant class. With the subacute and chronic cases the present paper does not deal. The affection now under consideration, as described in the opening paragraph, may be (1) bilateral from the onset, (2) monolateral, which is perhaps the most frequent manifestation, or (3) bilateral after an interval, that is to say there may be a shorter or longer period of time between the onset of the affection in the first and in the second eye, and it is with this third class of cases that I will deal and call attention, if not to a new group, to an uncommon variety of this group, to wit: one in which the retro-ocular inflammation is preceded by an attack of peripheral facial paralysis, either upon the same or upon the opposite side.

In illustration the following cases are reported:

CASE I.—Miss J. S., aged 30, a seamstress, born in Russia, presented herself for treatment on May 9, 1898, upon the kind recommendation of Dr. Barcus.

History.—The patient's early clinical history is unimportant, so far as it could be ascertained; of the health of her parents nothing is known; her sister, whom I examined, was sound. Between two and three years before her visit, after exposure to a draft, she developed facial palsy of the right side which lasted between two and three weeks, so I am informed by Dr. Barcus, her attending physician, and disappeared under the influence of blistering, alteratives and electricity. After her recovery the patient pursued her ordinary vocation as seamstress with comfort, her eyes, which were decidedly astigmatic, having been provided with proper glasses. Four days before she

consulted me, after a period of moderate right-sided headache, the sight of the right eye began to fail rapidly. She denied menstrual difficulties, either at this time or at any previous time in her history, and gave no very clear account of exposure to cold or draft, although she was accustomed to sit near an open window while sewing. Knee-jerks and station normal; no tremor; there was no anesthesia and the intellection was good.

Examination.—The girl was rather pallid, but gave no evidence of organic disease.

The vision of the right eye equaled seeing the movements of the hand in the upper field. The disc was a vertical oval, of pallid hue; both sets of vessels were slightly diminished and there was a strong venous pulse and faint retinal veiling. There was distinct pain above the eye and on movement. The center of the visual field was occupied by a large scotoma.

The vision of the left eye, after the correction of a mixed astigmatism, was 6-6, the accommodation normal, and the ophthalmoscope showed a rather pallid disc, and vessels smaller than normal. There were no traces of the former facial palsy except twitching of the right orbicularis and of the muscles at the angle of the mouth on the right side.

The usual treatment for retrobulbar neuritis, for practically all of the classical signs of this condition were present, was instituted, and after a vigorous course of iodide of potassium, and bichloride of mercury, with counter-irritation to the temple, and later full doses of strychnine, the vision gradually improved, and by the 25th of July had risen to 6-12 in the right eye, or fully one-half of normal.

At this time confusion of vision began upon the left side, preceded as before by pain in the brow and tenderness in the orbit. The disc appeared paler upon the temporal side than at previous visits, the veins being about normal, but the arteries small. The vision sank to 6-12, the accommodation failed, but a positive scotoma could not be demonstrated. Under ascending doses of iodide of potassium the vision rapidly improved, and from that date until the present time there has been no relapse, and now normal vision is restored to each eye and the patient has returned to her occupation.

CASE II.—Miss Anna K., aged 20, consulted me on September 29, 1896, for relief from keratitis.

History.—The patient's parents are healthy Germans, and the girl herself gives no history of severe illness or constitutional complaint other than much discomfort during menstruation. In 1893 she had an attack of sclero-keratitis of the right eye and soon afterwards facial palsy of the right side, which lasted for six weeks and disappeared under the influence of al-

teratives and electricity. A second attack of sclero-keratitis occurred in 1895 and was unassociated with facial palsy. The present attack did not differ from the others.

Examination.—The girl is well formed, of good coloring, and with the exception of dysmenorrhea and ocular disturbances, in fair condition. The vision of the right eye was 6-60, and the eye contained the ordinary lesions of patchy opacity of the cornea following severe scleritis. In the left eye the vision was 6-5, the accommodation normal, the media clear, the optic disc was of good color and the retinal circulation normal. The eye rapidly improved under treatment.

About six weeks after her first visit, and without any special history of exposure to cold or draft, and with no ear disease, she appeared with right facial palsy, inability to close the eye or wrinkle the brow, etc., which never, however, became complete. There was gradual restoration of function, although the right side of the face continued to be rather stiff, with a good deal of twitching at the angle of the mouth.

The patient was seen from time to time, but did not require special treatment until June 16, 1898, when she reappeared with the history that four days previous to her visit, after sleeping in a draft and when the night had become suddenly chilly, she awakened with a blur before the *left* eye, which rapidly developed until at the date of her visit she had only faint light perception in the peripheral field, the entire center of which was occupied by an absolute scotoma. The ophthalmoscope revealed moderate congestion of the disc. She was at once admitted to the Polyclinic Hospital and placed upon a treatment which consisted at various times of mercurial inunctions, pilocarpine sweats, iodide of sodium and counter-irritation. In two weeks vision was 3-60, excentric. The optic disc had lost its congestion, but there was decided pallor in the lower and outer quadrant and the ophthalmoscope revealed a large scotoma breaking through below. From that time until the present the condition has remained much the same, although the vision of the left eye has decidedly improved, and at the last examination, 10, 24, 98, was 6-50 when the head was rotated slightly to the left.

On August 9 I sent the patient to Dr. John H. W. Rhein, in order that he might examine the case electrically. He reports as follows: "I find quantitative changes only, in slight degree, in the muscles of the affected side of the face, reaction of degeneration cannot be obtained, and the nerve transmits the currents well, though not so well as on the unaffected side." A careful application of galvanism to the face improved its appearance; similar application to the eye was not followed by improvement. There were no signs of central nervous

disease; knee-jerks normal; station good; no patches of anesthesia; no tremor, nor change in voice, and no defect of intellect.

In *résumé* we find the first patient had, first, right facial palsy; two years later right retrobulbar neuritis, followed two and one-half months later by a mild left retrobulbar neuritis; ultimate complete recovery. The second patient had, first, right sclero-keratitis, probably depending on menstrual disturbance (whether there was retrobulbar neuritis at this time cannot be ascertained, but probably not); soon after right facial palsy; two years later again sclero-keratitis, without facial palsy; one year later sclero-keratitis, and six weeks later right facial palsy; finally, two years later, *left* retrobulbar neuritis, terminating in partial atrophy of the optic nerve. Omitting the attacks of sclero-keratitis, which probably have a different etiology, the patient had two attacks of right facial palsy, followed two years later by a severe left retrobulbar neuritis.

Naturally, there is no reason why a similar cause operating should not at one time attack the optic nerve as it passes through the optical canal and at another the facial nerve in the Fallopian canal, or, to quote the words of Mr. Marcus Gunn,² "Cases of retro-ocular inflammation of the optic nerve may be compared with those numerous instances of inflammation of the portio dura in its bony canal, many of which are attributed to cold or rheumatic inflammation of the sheath." It would seem, however, that not only may these affections be compared, but also that they may occur in the same subject, in a certain sense one affection replacing the other, or, perhaps, more accurately, the same cause operative in the same susceptible subject may produce either neuritis in the Fallopian canal, or neuritis in the optical canal. In other words, the subject of common facial paralysis due to neuritis again exposed to the original cause of the palsy may develop, not a recurrence of the Fallopian neuritis, but a neuritis in the optical canal.

With reference to the pathological lesions found in cases of retrobulbar neuritis, the investigations of Elschnig and others indicate that there is an interstitial neuritis with secondary degeneration of the nerve fibers. It is probable that a perineuritis, or a peri-osteitis in the optical canal would produce the

² Trans. Ophth. Soc. of the U. K., Vol. XVII, 1897, p. 118.

same symptoms, and as Gifford suggests,³ it is not likely that a pachymeningitis spreading into the optical canals would be followed by the ophthalmoscopic appearances and clinical signs already described as characteristic of this disease.

That a peripheral facial paralysis may be caused by a lesion of the nerve in the Fallopian canal was demonstrated by May's case of leucocythemia with facial paralysis, caused by an infiltration of lymphoid cells and destruction of the nerve fibers in the Fallopian canal. Adolf Meyer⁴ found in a case of facial palsy of ten days' duration, occurring in a paretic dement, hemorrhagic infiltration of the periosteum of the internal auditory canal and changes of reaction to a peripheral lesion in the facial nucleus of the same side. Dejerine and Theohari⁵ report a case of peripheral facial paralysis, of the so-called rheumatic type, in which they found considerable lesions of parenchymatous neuritis in all of the terminal branches of the facial, but especially in the inferior facial. The degeneration was less intense in the intra-petrous portion of the nerve.

In the more chronic types of retrobulbar neuritis, producing the clinical symptoms ordinarily known as central amblyopia, we are accustomed to describe the histological changes as an interstitial sclerosing inflammation, which Sarnelsohn compared with the same pathological process visible in interstitial hepatitis, but it has been well stated that the descriptions of the optic nerves in the recorded cases agree quite as well with an inflammation of the connective tissues of the nerve causing secondary atrophy of the nerve fibers, as with an atrophy of the latter, associated with secondary interstitial changes following in its track.

³ An American Textbook of Diseases of the Eye, Ear, Nose and Throat, p. 441.

⁴ Journal of Experimental Medicine, Vol. II, No. 6, 1897.

⁵ Comptes rendus de la Société de Biologie, 1897, p. 1033.

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83. TRIGGER FINGER ASSOCIATED WITH ACROPARESTHESIA. Riesman (Philadelphia Polyclinic, 7, 1898, p. 163).

A woman of 49 years who had had two previous attacks of numbness and "pins and needles feeling" in the right hand and arm, became afflicted a third time with the same trouble, and to it was soon added a frequent locking of the little finger of the affected extremity. No relief was afforded by potassium iodide and faradism, but on ergot, as recommended by Sinkler, the paresthesia as well as the peculiar catching of the finger rapidly disappeared.

PATRICK.

ON THE RELATIONS OF INFANTILE SPINAL PARALYSIS
TO SPINAL DISEASES OF LATER LIFE.*

BY WILLIAM HIRSCH, M.D.

The fact that infantile spinal paralysis may give rise to spinal diseases in later life, as long as from thirty to fifty years after the occurrence of the acute poliomyelitis anterior was first recognized by a number of French authors. Ballet, Dutil, E. Thomas, Sattler, and Charcot have reported cases of this kind, and recently similar cases have been published by M. Laehr, A. Dutil, Fibroy, and Langer. Altogether, nine cases are recorded. An autopsy has not been made in any case. As to the clinical symptoms, they indicate in all cases an exclusive affection of the motor neurons, sensory symptoms having been entirely absent, so that in every case the diagnosis of progressive muscular atrophy, mostly of the type of Aran-Duchenne, could be made. The only exception to this is in the case of Laehr, in which the patient complained of pains in the knee-joint and shoulder-joint, and also had difficulty in urinating. The latter symptom, however, Laehr refers to a hypertrophy of the prostatic gland; and the pain in the joints is not a very rare symptom of progressive muscular atrophy, so that according to its clinical aspect, this case also could be placed in the same category.

There is, however, an interesting difference among these cases as to their relation to the previous infantile paralysis. In some cases (Dutil, Thomas, Charcot, and Laehr), the muscular atrophy began in parts which had not been affected by the previous disease, but were even anatomically quite remote from the previously affected parts. In Charcot's case, for instance, the infantile paralysis affected the lower extremities, mostly the left leg, while the later disease began in the muscles of the right shoulder. In other cases the atrophy of the later disease started in the previously affected parts as well as in the healthy ones. In one case of Dutil, for instance, the poliomyelitis had affected the right arm and left leg, while thirty-six years later the muscular atrophy appeared in both upper and

* Read before the New York Neurological Society, Feb. 7, 1899.
For discussion on this paper see page 238.

lower extremities. The third class of cases seems to be represented by Langer's case, in which the poliomyelitis had affected both right extremities and a slight portion of the left leg, and in which the later disease was strictly confined to the muscles which had been attacked previously by the infantile paralysis.

Various theories have been offered as to the nosological relation between the early infantile paralysis and the later disease. According to some authors, irritation of the cord by the old lesion enfeebled this organ and made it a *locus minoris resistentiae*, which on any occasion might become susceptible to further disease. Charcot expressed the view that there existed in some individuals a certain disposition, a kind of vulnerability of the ganglionic cells of the anterior horns, which at some period of life might give rise to an acute poliomyelitis anterior, and at another to a progressive muscular atrophy, so that in fact both diseases would form different periods of one and the same pathological process. The third view was that the old scar, which was produced by the inflammatory process in the gray matter of the anterior horns, formed a latent but permanent inflammatory focus, which at any time might light up afresh and cause a new set of symptoms. Besides Charcot, Strümpell and others, Ballet and Dutil offered this theory, which they expressed in the title of their pamphlet: "Des quelques accidents spinaux déterminés par la présence dans la moelle d'un ancien foyer de myélite infantile."

It is evident that in view of the difference of the clinical aspect of these cases which I described above, any of these theories might hold good for some particular case. This, however, could only be ascertained by a post-mortem examination, which, so far as I know, has not yet been made in any of these cases. The following is a case in which I was fortunate enough to get an autopsy:

M. D., a tailor, forty-five years old, presented himself at my clinic in October, 1895. He denied having had syphilis. There were no nervous or mental diseases in his family. He was married twice; neither of his wives had ever had an abortion. He had two healthy children. He was in perfect health up to three years ago, when he first noticed a stiffness and weakness in his left leg, which gradually became worse. After some time the right leg also became affected. He soon became

unable to use his legs in working his sewing machine, and his gait became so much impaired that he was not able to stand or walk for any length of time. Besides this affection of the lower extremities the patient had nothing to complain of at that time. He stated that the use of the upper extremities was not impaired. There were no sensory symptoms whatsoever, nor was there a disturbance in the functions of the bladder and rectum. His sexual power was not diminished.

The objective examination revealed an atrophic condition of some of the muscles of the trunk and the upper extremities. The deltoid, the supraspinatus and infraspinatus of the right side showed quite a marked atrophy. There was fibrillation in the muscles of the trunk and shoulder. There was also diminished electric excitability, and in some muscles (deltoid and supraspinatus) reaction of degeneration. There was no atrophy in the muscles of the lower extremities, and the electric reaction was perfectly normal in these parts. The tendon reflexes of the lower extremities were considerably increased. The knee-jerks were extremely exaggerated, and there was bilateral ankle-clonus. The latter very frequently came on spontaneously, to the great annoyance of the patient. The tendon-of-Achilles reflex was increased. The superficial reflexes, like the cremaster, the abdominal and plantar, were present. The reflexes of the upper extremities, of the triceps, were not increased. Sensation to touch was perfectly normal in all parts of the body, as well as the sense of pain and temperature. The muscular sense was not impaired. There were no abnormal conditions in the functions of the cranial nerves, except that the tongue was slightly atrophied, and there was a marked masseter-reflex. Speech was in no way affected. There was no nystagmus. The reaction of the pupils, both to light and accommodation, was normal. The ophthalmoscopic examination revealed nothing abnormal.

Apart from this condition, there existed a very marked atrophy of the muscles of the left shoulder and upper arm. The deltoid had almost entirely disappeared. The biceps and triceps were reduced to a minimum, and the supraspinatus and infraspinatus, as well as the pectoral muscles, were also atrophic in the highest degree. He could, however, perform all motions with this extremity, though in a less degree.

When the patient came to me he did not mention this affection, because it had existed, as he said, as long as he could remember, since his earliest childhood. From the present state and from the history of this affection, as it could be learned through his relatives, it became evident that as a child of about two years he had an attack of infantile spinal paralysis, leaving behind the affection of the left upper extremity.

In this condition I presented the patient to the New York Neurological Society at the meeting of October 1, 1895. I maintained at that time that it could be proved by the history of the clinical symptoms that the pathological process of the later disease started from the place where the old scar of the poliomyelitis anterior was located. This lesion lay apparently in the left anterior horn of the cervical region of the cord. I stated at that time that as the man had first noticed weakness and stiffness of the left leg, the process must have approached first the left pyramidal tract which lay next to the scar, then after the affection of the right horn, which showed itself in atrophy of the muscles of the right upper extremity, the pathological process must have extended to the right pyramidal tract, causing a spastic condition in the right leg. According to this, we had an affection of both anterior horns and both pyramidal tracts in the cervical region of the cord, and, therefore, I called the case one of amyotrophic lateral sclerosis.

The further development of the clinical symptoms was as follows: The spastic condition of the lower extremities became gradually more and more intense, so that after three months, in January, 1896, the man was not able to walk at all. At this time he began to complain of very severe shooting pains in both lower extremities, which gradually became so intense that he had to be given hypodermic injections of morphine at frequent intervals. From the month of March he was confined to his bed. He was no longer able to sit up in bed or even to raise his head. Both lower extremities were in a permanent position of flexion and adduction. The spastic adduction was so intense that he had to wear a cushion between the two knees in order to overcome the severe pressure. The rigidity of the limbs was such that they could not even forcibly be abducted or extended. The shooting pains grew worse and worse, while the atrophy of the muscles of the lower extremities made no further progress during the last six months. The paralysis of both upper extremities, however, became gradually worse until the patient could not move the left extremity at all, with the exception of some slight flexion of the fingers. On the right side the arm was also completely paralyzed, but the movements of the hand seemed unimpaired during the whole time of his disease. Sensation was perfectly normal. The lower extremities were even somewhat hypersensitive. The function of the bladder and rectum had remained normal during the whole time.

In April he began to experience some difficulty in swallow-

ing, which gradually became more marked until towards the end of May he had become unable to swallow at all, so that he had to be fed with the tube. Without developing any other symptoms he died on the 14th of June, 1897.

A post-mortem examination was made about eight hours after death. On account of the great difficulties under which the autopsy had to be made, the internal organs and the peripheral nerves could not be examined. Only the brain and the spinal cord were obtained. The skull was symmetrical and showed no abnormalities. There were no adhesions between

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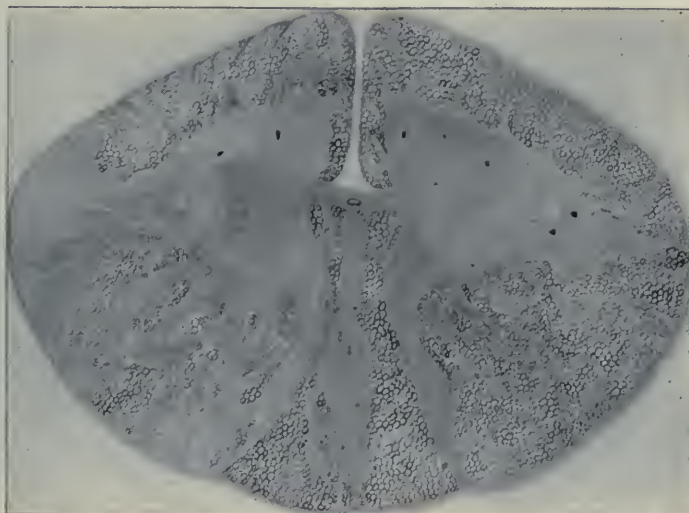


FIG. I. Cross-section through the second cervical segment.

it and the dura. The sinuses were filled with blood. The pia appeared hyperemic, but could be removed from the cortex without difficulty. The convexity of the hemispheres was symmetrical. The central convolutions showed no difference in size on both sides. Cross-sections through the medulla oblongata revealed macroscopically nothing abnormal. The spinal cord, however, showed indistinct outlines of the gray substance at various places. The consistency was normal, while the color seemed to be a little more reddish than under normal circumstances. Brain, medulla and cord were hardened in a 5 per cent. formalin solution, and small pieces were imbedded in paraffine and serial sections made through the entire cord and medulla oblongata. Parts of all convolutions of the cerebrum and the subcortical ganglia have been examined.

For practical reasons I will describe the microscopical condition of the spinal cord from above downward, beginning just below the cervico-medullary junction.

Second cervical segment. The left anterior horn shows a marked shrinkage, the lateral diameter being half that of the right horn. There are no ganglionic cells in the left horn at all, except a very few highly atrophied cells at the region of Stilling's nucleus. On the right side the different groups of ganglionic cells are all present, but also in a condition of atrophy. They are all considerably shrunken and in a chromo-

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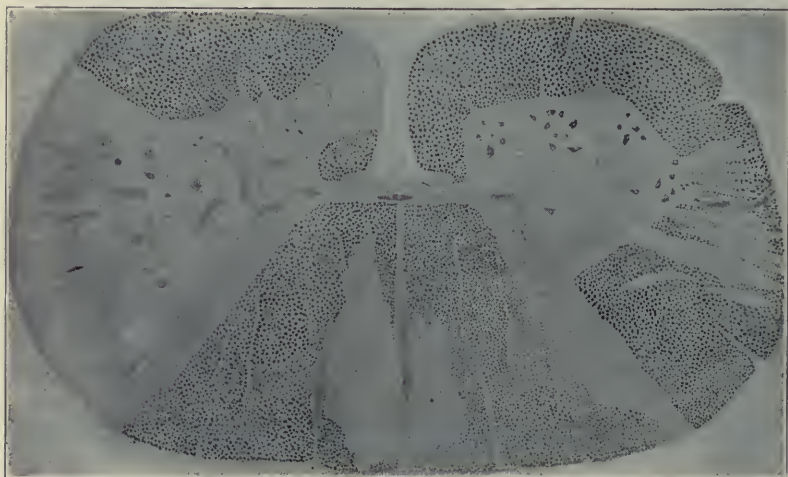


FIG. II. Cross-section through the sixth cervical segment.

philic condition. The pericellular spaces are enlarged. There is a proliferation of blood-vessels in the gray substance, and the perivascular spaces are considerably enlarged. The walls of the blood-vessels, however, are of their normal thickness and show no changes whatsoever.

The white substance is normal with the exception of the columns of Goll, where there is an irregular field of proliferating connective tissue extending from the center between the gray matter and the pia towards the periphery.

Third and fourth cervical segments.—There is a proliferation of connective tissue starting from the left anterior horn, extending irregularly into the white substance. Otherwise the conditions are the same as in the second cervical segment.

Fifth cervical segment.—The left anterior horn is still considerably smaller than the right one. The gray substance is found to be very vascular, and there are a great many granular cells scattered about, while the left horn has only very few highly atrophied cells belonging to the medial group. The cells of the right horn show only slight changes. They are of about normal size, but are mostly homogeneous. Nissl's granules have evidently disappeared. The axis-cylinder is broken off in some cells, and in other instances they appear to be swollen. The proliferation of connective tissue starting from

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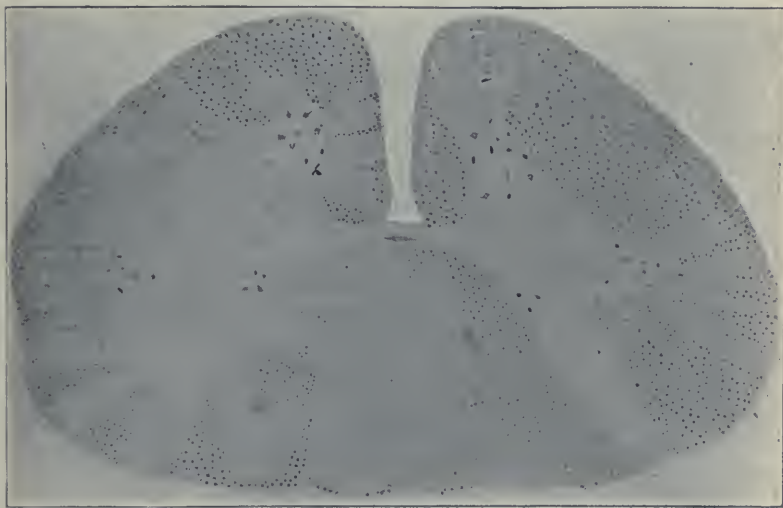


FIG. III. Cross-section through the first dorsal segment.

the left horn into the white substance is more marked than in the previous segment. The area of connective tissue in the columns of Goll is also the same as before, and contains many blood-vessels.

Sixth cervical segment.—The right horn contains the different groups of ganglionic cells, but they are nearly all atrophied. A good many cells contain a considerable amount of pigment. The proliferation of connective tissue from the left horn has spread fan-like through the entire white substance to the periphery, destroying the white substance altogether. A smaller fan-like proliferation spreads from the internal anterior corner of the left horn through the anterior columns of the white matter. On the right side there is a smaller prolifera-

tion of connective tissue starting from the right anterior horn, so that both horns give the appearance of a lateral enlargement. The blood-vessels appear very numerous in both the gray and white substance. They are full of blood, and the perivascular spaces are considerably enlarged. The posterior columns show a large area of connective tissue covering nearly the entire dorsal half of the columns of Goll. Here we also have the same

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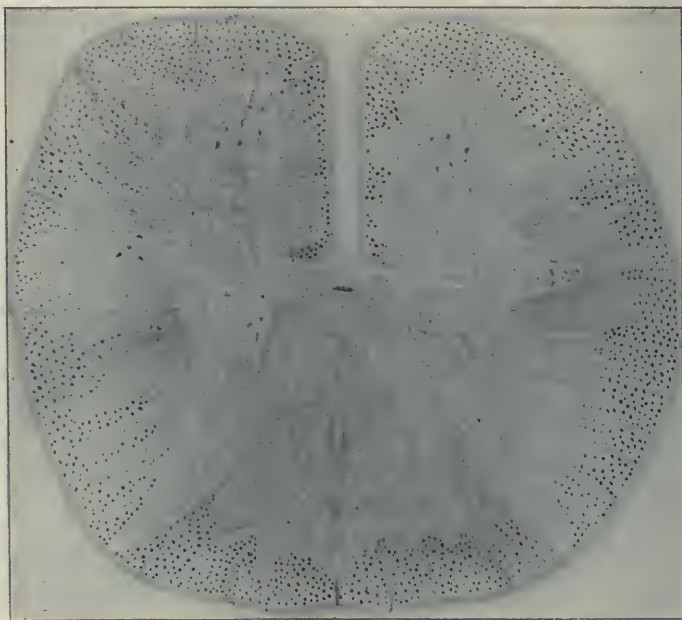


FIG. IV. Cross-section through the seventh dorsal segment.

proliferation of blood-vessels, around which there are many granular cells.

Seventh cervical segment.—There are very few ganglionic cells in the left horn, and those in the right horn are all atrophic. The fan-like proliferation of connective tissue from both horns has become larger. Of the posterior columns, those of Goll are entirely converted into connective tissue, while those of Burdach look more normal, otherwise the same as before. There are no normal cells in either horn, except a few in the lateral group of the right side amidst the proliferating connective tissue. On both sides there is a proliferation of connective

tissue from the anterior internal corner of the anterior horn through the white substance to the periphery, otherwise the same as before.

Eighth cervical segment.—On the right side all the different groups of ganglionic cells are present, and they are perfectly normal; only a few show a slight atrophy. On the left side there are more atrophic cells than on the right, but still there are some normal cells here, too, and the lateral group especially

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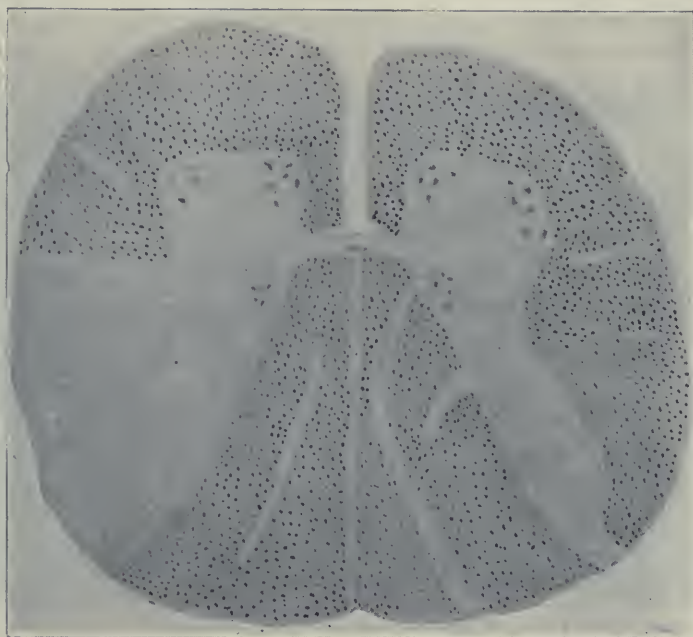


FIG. V. Cross-section through the second lumbar segment.

seems to be normal. We find here some normal cells within the fan-like expansion of connective tissue. The atrophic cells all contain a considerable amount of granular pigment. The blood-vessels are enormously enlarged and increased in number. But nowhere are there any changes in their walls.

First dorsal segment.—The ganglionic cells on both sides in the different groups in the anterior horns and in the columns of Clarke, are mostly atrophic; only here and there we find some normal cells. The fan-like proliferation of connective tissue on the left side embraces the entire half of the cord, with the exception of a small area of the anterior white column. On

the right side, this proliferation has become less, so that we have considerably more normal white fibers. The posterior columns are converted into connective tissue, with the exception of the larger part of the right column of Burdach.

From the second to the sixth dorsal segment we have about the same condition as in the first dorsal segment, the normal ganglionic cells alternating with highly atrophic ones, and the expansion of the areas of the proliferating connective tissue in the white substance varying on the different levels. The proliferation of blood-vessels remains very considerable, and also

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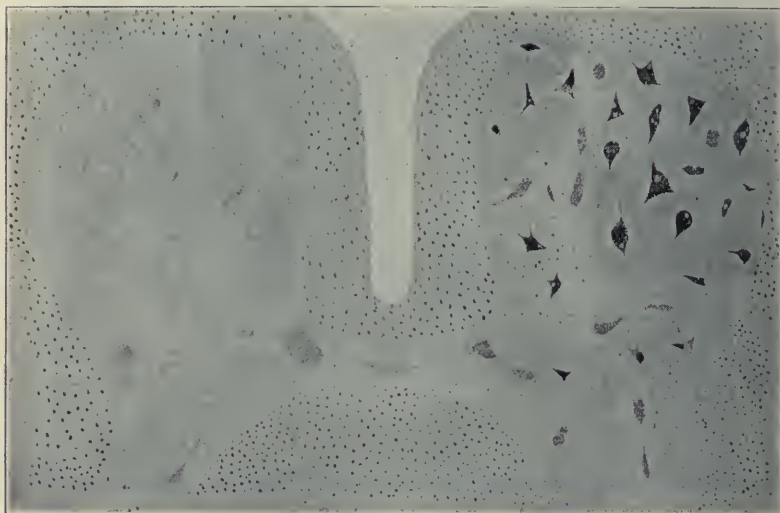


FIG. VI. Cross-section through the fourth lumbar segment.

the enlargement of the perivascular spaces. At some parts we find a considerable accumulation of granular cells.

Seventh dorsal segment.—We find nearly the entire cord converted into connective tissue, so that it has an aspect of a transverse myelitis, only at the periphery all around the section there are normal nerve fibers; the outlines of the gray substance being difficult to recognize. The degree of atrophy of the nerve cells seems not to be in proportion to the pathological process of the surrounding tissues. We find sometimes perfectly normal cells surrounded by connective tissue.

The lower we go down in the cord the larger grows the normal space at the periphery.

Ninth dorsal segment.—The white substance has again be-

come normal with the exception of a small portion at the left side, where a fan-like proliferation of connective tissue extends again from the anterior horn to the periphery. The columns of Goll also are converted into connective tissue. The gray substance is destroyed nearly altogether, and only a very few cells are present.

Tenth dorsal segment.—Again the entire cord is converted into connective tissue, with the exception of a small part of the periphery.

Eleventh dorsal segment.—There are very few cells in both anterior horns, while the lateral horns and the columns of Clarke are nearly normal. The white substance has become more normal again, especially on the right side, while the left side is, however, destroyed, and the posterior columns show nearly normal fibers at the lateral margins.

Twelfth dorsal segment.—The proliferation from both horns is increased again, so that on both sides the white columns appear to be destroyed. There are only normal white fibers in the anterior columns. The cells in the anterior horns and columns of Clarke are present, although highly atrophic.

First lumbar segment.—First lumbar segment shows the same conditions as the twelfth dorsal.

Second lumbar segment.—The white substance on both sides is again more normal, with the exception of a large fan-like proliferation of connective tissue on the left side.

Third lumbar segment.—On the right side the white substance seems to be entirely normal, while on the left side there is still a considerable area of connective tissue. The posterior columns have become normal. The cells are all atrophic. In the left horn the cells seem to have disappeared altogether.

Fourth lumbar segment.—The white substance has become perfectly normal, with the exception of a very small area on the left side. The ganglionic cells on the right side are large in size, but contain an unusual amount of pigment. A good many cells are chromophilic, and sometimes the axis-cylinder seems to be broken off. On the left side there are no cells at all, with the exception of a very few highly atrophied remnants. There are a good many empty spaces, indicating the disappearance of the cell. We find the same condition in the fifth lumbar segment.

Towards the lower part cells appear again on the left side. The white substance has remained normal. The sacral columns appear normal, with the exception of some atrophic cells.

In the first sections of the medulla oblongata we find the same proliferation of connective tissue in the ventral part, affecting mostly the pyramidal tracts, sometimes extending dorsad into the lemniscus and the substantia reticularis alba. The nucleus of the hypoglossus contains many atrophic cells

on both sides. On the left side the proliferation of connective tissue occupies a field which is limited dorsad by the substantia gelatinosa and the nucleus of the hypoglossus; laterally by the nucleus lateralis. Besides the nucleus of the hypoglossus, those of the glossopharyngeus and vagus and the nucleus ambiguus contain many atrophic cells, while these nuclei on the right side are normal. At the level where the fibers of the glossopharyngeus begin to leave the medulla, everything has become normal, and remained so up to the level of the nucleus of the oculomotorius. The only changes are enlargement of the perivascular spaces, not only in the medulla oblongata, but also in all parts of the brain. The cell layers of the cortex, over all parts of the brain have been carefully examined and have been found perfectly normal.

A number of interesting questions arise from these anatomical findings. The most important, of course, is the diagnosis. When I presented the patient to the Neurological Society three years ago, I had made the diagnosis amyotrophic lateral sclerosis, maintaining that the pathological process had started from the left anterior horn of the cervical part of the cord and proceeded from there, first to the left pyramidal tract, then to the right horn and right pyramidal tract. The microscopical examination shows this assumption to be true. A glance at the cervical part of the cord will show an anatomical condition of this kind. Still, are we justified in calling this a case of amyotrophic lateral sclerosis? The clinical course of the disease would surely be in favor of it. We had the typical three stages which Charcot described: the atrophy, the rigidity with the muscular contractures and the bulbar symptoms. The rapid course of the disease, which lasted only a few years, would also correspond to Charcot's description. The pain which the patient experienced towards the end of his life does not, of course, belong to the disease, but could be explained somehow or other by the muscular contractures or some complication. From the anatomical findings, however, the diagnosis of amyotrophic lateral sclerosis cannot be sustained. Whether amyotrophic lateral sclerosis is a *morbis sui generis*, which is to be distinguished from progressive muscular atrophy and bulbar paralysis, or whether, as many authors maintain, these diseases form only different stages or degrees of one and the same disease, is a question which cannot be discussed

here. Whatever view, however, we might hold regarding this question, one must regard amyotrophic lateral sclerosis, whether it forms a separate disease or not, as a systemic disease; like progressive muscular atrophy, a primary degeneration of the motor neurons.

To avoid misunderstanding, it would be still better to adopt Dana's suggestion and change the name systemic disease into neuron disease. The important question then in this special case would be: have we to deal with a neuron disease, that is, a primary degeneration of a certain group or groups of neurons belonging to one system, or with some other pathological process through which the neurons have become affected secondarily? There can be no doubt that we have to deal here with the latter condition. In favor of this view are:

First, the anatomical process itself. There is at several levels of the cord, but especially at the lower cervical and upper dorsal region, a fan-like proliferation of connective tissue from the gray substance, which we do not see in primary degeneration of the white columns. It seems evident that this proliferation of connective tissue has destroyed the white substance, but has not been produced through the degeneration of the latter, as is the case in primary neuron diseases. The enormous proliferation of blood-vessels, as well as the considerable enlargement of the perivascular spaces, do not indicate a primary degeneration of the neurons.

Secondly, the topography of the pathological process. The inflammatory process which apparently originated in the gray substance, spread in all directions and involved all parts of the white substance without any regard to systems. We find the columns of Burdach and Goll and the anterior columns affected, as well as the pyramidal tracts; that the inflammation did not follow any systemic paths, but spread from a central focus in all directions, is especially evident in the dorsal region, where the outlines of the gray substance have become very indistinct and the inflammatory field occupies nearly the whole cord, leaving some normal fibers only around the periphery, regardless of the system to which they belong.

Thirdly, The atrophy of the ganglionic cells is not in proportion to the changes of the surrounding tissue. We find

sometimes normal or only slightly atrophied cells amid the most remarkable changes, so that the affection of the nerve cells must be regarded as a secondary matter.

Therefore, we have to deal here with a case of interstitial myelitis, probably of vascular origin, in which the nerve cells have become atrophied secondarily.

As to the nature of systemic or neuron diseases and their relation to other inflammatory processes of the central nervous system, there seem to be some discrepancies among different authors. There is on the one hand a tendency to deny the existence of systemic diseases altogether. The supporters of this view maintain that all systemic diseases, including tabes and progressive muscular atrophy, consist of myelitic processes by which the neurons are affected secondarily. On the other hand, there are some followers of the theory of systemic diseases who draw very narrow limits to the interstitial myelitic processes, considering, for instance, poliomyelitis anterior a systemic disease, as was first suggested by Charcot.

Recent anatomical investigations of fresh cases of infantile spinal paralysis, as well as the generally accepted theory of its infectious nature, have put it, I think, beyond doubt that we have to deal here, not with a systemic disease, but an interstitial inflammatory process. The fact that the anterior horns are always the main seat of the affection is due to the more abundant blood supply of these parts. The statement of some authors that certain groups of ganglionic cells are generally more affected by the disease than others has not been confirmed by closer investigation. On serial section, some groups appear to be affected at some level and intact at another. The atrophy of the cells is dependent upon the distribution of the smaller branches of the central vessels, and the fact that in one section some cells are atrophic, while others are intact, is due to the difference in the blood supply, the different groups of cells belonging to different areas of blood-vessels.

As far as spinal diseases of later life are concerned, we must admit that in many cases it is not only extremely difficult, but sometimes quite impossible, to distinguish between a systemic or neuron disease and a myelitis merely from the clinical symptoms. My case is a good illustration of this fact. Every-

body who saw the case agreed that it was a systemic disease; that is, either progressive muscular atrophy or amyotrophic lateral sclerosis, while the microscopical examination revealed quite different conditions. A myelitis might possibly simulate any neuron disease.

I am inclined to believe that quite a number of the cases which have been reported as having developed from an infantile spinal paralysis to a progressive muscular atrophy, after a microscopical examination, would prove to be, like my case, instances of myelitis, instead of primary neuron disease.

The next question would be whether this man's infantile spinal paralysis had any relation to his later disease, and if so, of what nature was this relation? I have mentioned the different theories which have been advanced on this subject. Most of them are based on the theory that poliomyelitis anterior is a systemic disease and could not therefore be applied to this case.

The third theory, however, would correspond to the clinical and anatomical condition of this case; that is, that the old scar which was produced by the inflammatory process in the gray matter of the anterior horns, formed a latent but permanent inflammatory focus, which at any time might light up afresh and cause a new set of symptoms. That in my case the old scar in the left anterior horn of the cervical part of the cord formed the starting point for the disease was diagnosed during life, and has been confirmed by the autopsy. Whether the old scar contained the poison to set up the new disease, or whether we have to deal with a new infection which attacked the old scar first, as the *locus minoris resistentiæ*, is, of course, a question which cannot be decided with absolute certainty. I think, however, that there is no necessity of assuming a new infection. It is perfectly possible that, as Strümpell says, the virus in the old scar behaves like an encapsuled tubercular focus, remaining dormant for a long time, and then setting up a new inflammation.

Another point of interest is whether this case could contribute to our knowledge regarding the functions of the cells of the spinal cord. Cases of infantile spinal paralysis, which have left behind atrophies of isolated muscles, and which come

to autopsy in later life, are especially appropriate for this study; in fact, they equal in many respects an experiment. Naturally, such cases are very rare. Charcot reports that he saw the scar of infantile spinal paralysis in the anterior horns as long as sixty years after the disease, but a special study of the cells as to their function was not made by him. A very valuable contribution to this part of science has been furnished by Collins, who had the chance of examining a spinal cord of a man who died from a nephritis, and who had an attack of infantile spinal paralysis twenty-three years previously. Collins was able to corroborate by his case the views regarding the nucleus of the upper extremity. This nucleus is composed of three groups of cells located in the lateral part of the anterior horns, and extending from the lower part of the fourth cervical into the first dorsal segment. Collins was furthermore enabled to show by his case that a group of cells belonging to this upper-extremity nucleus, being located at the seventh cervical segment and being arranged internally, governed the function of extending the fingers; while another group of cells between the last cervical and first dorsal segments, being arranged laterally, is concerned in supplying the muscles that cause flexion of the fingers.

My case is not quite as appropriate for this study, because the man developed another spinal disease, which made it rather difficult to distinguish between the old and recent affection, while Collins' patient exhibited the spinal cord of the old infantile paralysis without any further changes. There are, however, some valuable facts which can be utilized in spite of this. In order to compare the anatomical findings with the clinical symptoms, I may be allowed to recall with a few words the progress of muscular disease in the patient's upper extremities.

His left arm remained in a highly atrophied condition after the infantile disease. The muscles of the shoulder and upper arm were involved most. The muscles of the hand had remained perfectly normal. During the course of the later disease the whole left extremity became gradually paralyzed, the only movement which the man could perform being a slight flexion of the fingers. The right upper extremity, which had

not been affected at all by the infantile paralysis, also became paralyzed during the later disease, but here only the movement of the arm had been lost; the hand could be moved perfectly well in spite of a considerable atrophy of the interossei. The microscopical examination revealed an entire destruction of the upper-extremity nucleus on the left side, with the exception of a few cells at the outer margin of the left anterior horn in the eighth cervical segment. If we hold these cells responsible for the limited movement of flexion which the man could perform, we can corroborate Collins' statement as to his outer group in this respect.

On the right side the upper-extremity nucleus was destroyed down to the middle part of the seventh cervical segment. At this level the cells of this nucleus appeared normal, with the exception of the anterior group, which was missing. With the beginning of the eighth segment, however, this group also became normal, so that the entire nucleus appeared in good condition down to the beginning of the first dorsal segment. This is in perfect harmony with the unimpaired movements of the man's right hand, the atrophy of the interossei being due to the atrophy of the cells in the first dorsal segment.

The atrophy of the cells which innervate the muscles of the back, *i. e.*, the group of cells which we located internally in the anterior horns, accounts for the man's inability to sit up or hold up his head.

There seems to be a discrepancy in the findings of the cortical cells between this case and the one of Collins. While in my case I could not detect any abnormal conditions in the cortical areas corresponding to the part which was affected by the infantile paralysis, Collins reported an atrophy both in size and constituents of this area. This, however, is due to the fact that my patient, in spite of the atrophy, could move and use his extremity, while in Collins' case the affected extremity was perfectly motionless. This difference between the two cases would, therefore, corroborate Collins' theory, that not the atrophy but the inactivity produces the affection of the other group of neurons. The fact that in my case the cortical cells of the cortico-spinal neurons were perfectly normal, in

spite of the severe affection of the pyramidal tracts, is another support of the statement that we have to deal in this case, not with a systemic or neuron disease, but with a local inflammatory process, *i. e.*, a myelitis.

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84. EINE NEUE FÄRBUNG FÜR DAS NERVENSYSTEM (A New Stain for Nervous Tissue). P. Kronthal (Neurologisches Centralblatt, 18, 18 9, p. 196).

Kronthal has described a new method of treating nervous tissue which permits of the study of the nerve cells as well as the fibers. It combines the method of Golgi and that of Weigert in specimens which are successfully treated. He first hardens the nervous tissue, perfectly fresh, in a mixture consisting of equal parts of a saturated aqueous solution of the formate of lead and 10 per cent formalin. The pieces remain in this fluid five days at the ordinary temperature. They are then transferred, without washing, into a mixture of equal parts of 10 per cent. formalin and hydrogen sulphide solution (a saturated solution of sulphureted hydrogen in water), in which mixture they remain five days. They are then washed, dehydrated in alcohol, imbedded in celloidin, and the sections cleared in xylol-carbolic solution and mounted in balsam with cover. According to the author's experience, they suffer no change after at least ten months.

For the conservation and staining of an entire brain, the author has tried the following procedure: The brain was placed for 14 days in $2\frac{1}{2}$ liters of 1 per cent. formalin and saturated solution of formate of lead equal parts of each; and then in $2\frac{1}{2}$ liters of 10 per cent. formalin and $2\frac{1}{2}$ liters of hydrogen sulphide solution, and allowed to remain in this for 14 days. It was then kept in a solution of equal parts of glycerine and 90 per cent. alcohol until it was ready for sectioning. The glycerine, while making the brain tissue a little softer, makes it more elastic. Brains kept in alcohol alone are too brittle for large sections.

JELLIFFE.

LIPOMA OF THE FILUM TERMINALE.¹

BY WILLIAM G. SPILLER, M.D.,

PROFESSOR OF DISEASES OF THE NERVOUS SYSTEM IN THE PHILADELPHIA POLYCLINIC, ASSOCIATE IN THE WILLIAM PEPPER LABORATORY OF CLINICAL MEDICINE, UNIVERSITY OF PENNSYLVANIA.

Tumors of the filum terminale are, as a rule, small, as the structure from which they grow is a narrow band composed chiefly of the tissue forming the continuation of the pia. When these growths attain large size they involve the lower lumbar and sacral roots forming the cauda equina, and we speak of them then as tumors of the cauda equina, though it by no means follows that every tumor of the cauda equina originates in the filum. Tumors of the filum terminale are exceedingly rare, and without doubt are occasionally overlooked. The filum is enveloped in a thick mass of anterior and posterior root fibers, and is seen only when these fibers are separated. This examination is not usually made, and malformations of the filum are not likely to be detected unless special attention is directed to this portion of the cord.

A little more than three years ago, while in the laboratory of Dr. Dejerine, I studied the structure of the conus medullaris in quite a large number of cases of tabes dorsalis, and in the spinal cord of one of these cases a tumor of the filum was found, about half a centimeter below the union of the filum with the conus. The tumor formed a distinct uniform swelling of the filum (Fig. 1), and in transverse section was considerably larger than the smallest transverse section of the conus showing the formation of the spinal cord in central gray and peripheral white matter. The interesting specimens have been in my possession nearly four years, but have never been reported. I am indebted to Dr. Dejerine for the material, and for the permission to publish the findings.

The case was one of tabes dorsalis, and the tabetic degeneration of the posterior roots and posterior columns is independent of the tumor of the filum. Even in low sacral sections the posterior columns are distinctly degenerated.

¹ From the Salpêtrière, Paris (laboratory of Dr. Dejerine), and the William Pepper Laboratory of Clinical Medicine.

Read before the Pathological Society of Philadelphia, Feb. 23, 1899.

The tumor is a lipoma. On one side of the growth the structure of the filum may be distinctly seen. Within the filum is a mass of glear cells which represent the termination of the cell-masses surrounding the central canal of the cord; and about these cells the connective tissue of the pia is found. These cells lie in a granular and somewhat fibrillar matrix. On three sides of the filum, and yet forming an integral part of it, is loose connective tissue resembling fatty tissue. The tumor has not a distinct unbroken capsule, but along its edge in many parts the tissue is thickened by fibrous bands. Nerve fibers, stained black by the Weigert-Pal hematoxylin method,



FIG. I. Lipoma of the filum terminale. The drawing has been made from memory. The width of the tumor is exactly that of the microscopical section. Inaccuracies, if they exist, are very unimportant.

are found in small groups here and there within the fatty tissue of the tumor, and probably represent the delicate nerve fibers that may be separated from the periphery of the filum in many normal cases. These nerve bundles, as well as the vessels, are surrounded by fibrous tissue and form small masses of fibrous tissue within the fatty tumor. The pia of the filum is infiltrated in its outer layers by fat cells (Fig. II.) and a few fat cells are seen within the inner layers of the pia, but no fat cells are found

interior to the pial sheath. The fat cells of the tumor are round or oval, such as are usually seen in adipose tissue. In the delicate fibrillar structure between the fat cells numerous oval or round nuclei are found—round probably because they are cut transversely. The walls of the small blood vessels within the tumor are not noticeably thickened. At one part of the periphery of the tumor is a nerve bundle containing a number of nerve cells similar to those of spinal



FIG. II. Transverse section of the lipoma. The filum is seen at *a* and is surrounded on three sides by the adipose tissue.

ganglia (Fig. III.). Müller² has recently described these cells in the lower roots of the cauda equina, within the dura, and not forming a part of any spinal ganglion.

Argutinsky³ describes the enlargement of the central canal in the conus of the newborn child and human fetus. The description of this enlargement, known as the ventriculus

² Müller, *Deutsche Zeitschrift für Nervenheilkunde*, Vol. XIV, p. 1.

³ Argutinsky, *Archiv für mikroskopische Anatomie und Entwicklungsgeschichte*, Vol. LII, No. 3, p. 501.

terminalis, is most interesting and suggestive in regard to the etiology of syringomyelia. Argutinsky thinks that the ventriculus terminalis is not a persisting embryonal central canal, but is the result of proliferation of the ventricular wall. In this he differs from all other writers. The subject does not concern us at this time, intensely interesting though it may be, but the statement that a very considerable accumulation of nuclei exists in the wall of the ventricle throughout its length



FIG. III. Section of a nerve root (represented at *b* in Fig. II) containing nerve cells of the form of those within the spinal ganglia.

is doubtless the explanation for the accumulation of nuclei we find in the filum terminale.

Lipoma of the central nervous system, especially the intradural variety, is one of the rarest of tumors. It has been observed a number of times in cases of spina bifida. Chiari⁴, for example, reported a case of spina bifida with lipoma involving the spinal membranes. In 1886 v. Recklinghausen⁵ reported

⁴ Chiari, Prager med. Wochenschrift, No. 50, 1884, p. 489.

⁵ v. Recklinghausen, Virchow's Archives, 105, 1886, p. 243.

a similar case with a myofibrolipoma surrounding the cord; the fatty mass was also found within the spinal dura. Bruns⁶, in his work on tumors of the nervous system, says that lipomata are not so rare in the lower thoracic and upper lumbar regions in association with spina bifida in children, and he reports a case of this kind. Ziegler⁷ and others make similar statements.

We can hardly be surprised that lipomata should have been observed when the lower part of the spinal column is congenitally deficient.

When we examine the literature for cases of intravertebral lipoma we find little to reward our search. Lipoma is rare within the cranial membranes, as well as within the vertebral. Ziegler, in his last edition, is contented with the statement that fibromata, lipomata and osteomata of the central nervous system are rare (l. c. p. 365). He makes no reference to the occurrence of lipomata within the spinal cord. In mentioning the tumors of the spinal meninges he puts lipomata last (l. c. pp. 308, 312).

Reisman⁸ exhibited before the Path. Soc. of Philadelphia a fibrolipoma of the infundibulum.

Oppenheim⁹, in his book on cerebral tumors published in 1897, says little is to be found in the literature concerning lipomata of the brain. In rare cases they have been found in the corpus callosum, in the corpora quadrigemina, within the ventricles, in the hypophysis, and a few other places.

The cases of lipomata within the vertebral column are so rare that I have made as thorough an examination as possible of the literature from the references I have been able to obtain. Schlesinger¹⁰ mentions probably all the cases reported, and, with three exceptions, I have been able to find and read all the original papers quoted by him. The journal in which one of these three cases appears is not correctly cited.

The earliest recorded cases are those of Chapelle and Albers.

⁶ Bruns, *Die Geschwülste des Nervensystems*.

⁷ Ziegler, *Lehrbuch der speciellen pathologischen Anatomie*, Ninth edition, Vol. II.

⁸ Reisman, *Proceedings of the Path. Soc. of Philadelphia*, Vol. I, No. 3, N. S., p. 64.

⁹ Oppenheim, *Die Geschwülste des Gehirns*, 1897, p. 14.

¹⁰ Schlesinger, *Beiträge zur Klinik der Rückenmarks und Wirbel-tumoren*.

In Chapelle's¹¹ case (1847) the spinal dura in a child of four years was surrounded by a large amount of fat from the cervical region to the end of the cord, especially on the posterior aspect. It was an extradural growth.

In the atlas by Albers¹², published also in 1847, the picture of a spinal cord is given with several lipomata on the outer side of the dura.

Obré,¹³ in 1852, reported a case in which a deposit of fat was found external to the theca, and between it and the bodies of the last cervical and first thoracic vertebra. It was a mass of fatty tissue, two and a half inches in length, the breadth of the canal and about half an inch in thickness, composed of the ordinary spherical fat cells. It did not differ from fat usually met with in other situations, excepting that the cells seemed to contain fat in a more solid and granular state. This deposit of fat pressed upon the cord.

In 1857 Athol Johnson¹⁴ reported a case in which a fatty tumor was found external to the spinal column and penetrating through an aperture large enough to admit the end of the finger in the posterior wall of the sacral canal. The tumor was dissected from the spinal membranes without any opening in the latter having been made.

Virchow¹⁵, in the same year, reported a case of multiple fatty tumors. One was found in the anterior cerebral fossa loosely connected with the arachnoid; another was found within the vertebral canal in the lumbar region, external to the dura. Virchow regarded this form of tumor as a peculiar variety of lipoma, and spoke of it as myxoma lipomatodes.

E. K. Hoffmann¹⁶ reported a case of gliomyxoma of the spinal cord with numerous fatty tumors upon the outer surface of the dura.

The case reported by Gowers¹⁷ in 1875 is the most inter-

¹¹ Chapelle, *Bulletins de la Société Anatomique de Paris*, 22, 1847, p. 6.

¹² Albers *Atlas der path. Anat. für praktische Aerzte*, Henry & Cohen, Bonn, 1847.

¹³ Obré, *Transactions of the Path. Soc. of London*, Vol. III, p. 248.

¹⁴ Johnson, *Transactions of the Path. Soc. of London*, Vol. VIII, p. 16.

¹⁵ Virchow, *Virchow's Archives*, XI, p. 281, 1857.

¹⁶ Hoffmann, *Zeitschrift für rationelle Medicin*, Vol. XXXIV, p. 188.

¹⁷ Gowers, *Transactions of the Path. Soc. of London*, Vol. XXVII, 1876.

esting in connection with my own that I have found. He observed a small fatty tumor attached to the *conus medullaris*, and apparently springing from the *pia*. The cord was from a patient who had had *tabes dorsalis*. The tumor measured half an inch from above down; half an inch from before back, and about three-eighths of an inch from side to side at its thickest part. This tumor contained striated muscular fibers, and was larger than the new growth in my case, for while the length was nearly the same, the transverse diameter of Gowers' tumor was greater. Like Gowers' tumor, mine is crescentic in transverse section, and surrounds one-half of the *filum* in the same manner as his surrounded the *conus medullaris*. Many of the nerve roots of the *cauda equina* were imbedded in Gowers' tumor, and mine contains many of the delicate filaments which lie close to the *filum terminale*. I did not have the opportunity of examining my tumor before it had been hardened in Müller's fluid, but in microscopical sections it has a very similar appearance to the one described by Gowers, except that the striated muscular fibers are absent.

Gowers does not think the tumor in his case was in causal relation to the degeneration of the posterior columns; neither can I hold this opinion in regard to my case, but it is, nevertheless, an interesting fact that the only two lipomata known of the *cauda* or *filum* were in tabetic patients.

Braubach¹⁸ reported the finding of a lipoma, 12 cm. long, in the arachnoid. The growth was adherent to the *dura* in certain parts, and the cord was much compressed.

Turner¹⁹ reported a case in which a lipomatous tumor was found in about the middle of the dorsal region of the cord, was about the size of a large filbert nut, and formed a well defined fusiform expansion of the substance of the cord at the affected part. The tumor was free from attachment to the external membranes of the cord, and the outer layer of *pia* sheathing it stripped off easily from a portion of it removed for microscopical examination. A transverse section of the tumor showed that the left half of it consisted wholly of adipose tissue, the cord appearing to have been pushed to one side. The cord,

¹⁸ Braubach, *Archiv für Psychiatrie*, Vol. XV, p. 489.

¹⁹ Turner, *Transactions of the Path Soc. of London*, Vol. XXXIX, p. 25.

however, was almost wholly destroyed by infiltration with a growth similar to that of the tumor.

Turner thought it might be supposed that a lipoma originated in a very obese subject, in the pia ensheathing the cord between the more superficial and the deep layers; that the mass compressed the cord, and that an infiltrating fibrous connective tissue growth became the seat of fatty infiltration. It seemed to him more probable that the growth, though having the appearance of a simple lipomatous tumor and consisting almost entirely of adipose tissue, was essentially of a sarcomatous nature, a lipomatous sarcoma or fibrosarcoma, originating in the pia and invading the cord. He seems to have been influenced in expressing the latter opinion by the extreme rarity of lipoma within the cord substance.

Témoin's²⁰ case was one of tumor in the lumbo-sacral region resembling spina bifida. The lipoma was connected with the tissues within the vertebral column by a pedicle.

Oustaniol²¹, in his thesis on tumors of the spinal membranes published in 1892, could find only five cases of lipoma of these membranes reported in the literature. These cases are cited in my paper.

Chipault refers to a case reported by Zavaleta and Masi²², in which a lipoma was found in the lumbar region and was connected by a pedicle with the arachnoid.

M. Allen Starr²³ reported a case in which a soft fatty tumor was found beneath the tenth thoracic vertebra; it extended across the posterior surface of the dura about half an inch from side to side, and was three-fourths of an inch in length. A second fatty tumor was found beneath the laminae of the eleventh thoracic vertebra.

The original reports of the cases of Holmes²⁴ (reference given by Schlesinger incorrect), Berenbruch²⁵ and Stümpell²⁶

²⁰ Témoin, *Archives provinciales de Chirurgie*, Vol. I, No. 2, 1892, p. 179.

²¹ Oustaniol, *Contribution a l'étude des tumeurs des méninges rachidiennes*, Paris, 1892, cited by Témoin.

²² Zavaleta and Masi, cited in *Études de chirurgie médullaire*, par A. Chipault, 1894, p. 346.

²³ Starr, *The American Journal of the Medical Sciences*, Vol. CIX, 1895, p. 622.

²⁴ Holmes, cited by Schlesinger.

²⁵ Berenbruch, cited by Schlesinger.

²⁶ Strümpell, cited by Schlesinger.

I have not been able to obtain. Schlesinger says that Holmes' tumor was extradural; Berenbruch's case was one of multiple angioliomata combined with an angioma of the spinal cord; Strümpell's was an intradural lipoma, but its location is not mentioned by Schlesinger.

These are the only cases of intradural or extradural spinal lipomata mentioned by Schlesinger, and the only ones to which I have been able to obtain references. Some cases may possibly have been overlooked and if so they have been overlooked also by all the writers to whom I have referred.

We find that from 1847 until 1899, a period of 52 years, only 16 cases of intradural and extradural spinal lipomata, without association with spina bifida, have been reported as occurring in man. Of these eleven were extradural (Chapelle, Albers, Obré, A. Johnson, Virchow, Témoin, E. K. Hoffmann, Zavaleta and Masi—tumor connected by pedicle with arachnoid—Starr, Holmes, Berenbruch), although one of these (Berenbruch) possibly has not been correctly placed, as I have not been able to obtain the original paper. Four of the sixteen (Gowers, Braubach, Stümpell, Spiller) were intradural, but not in the cord substance; and of these four only one was in the conus (Gowers) and only one in the filum (Spiller), unless Strümpell's tumor was situated in this region. One of the sixteen cases was a lipoma of the cord substance (Turner).

The large number of extradural in comparison with the intradural lipomata may probably be explained by the presence of the fat normally existing between the dura and the vertebrae.

While the tumor of the filum terminale in my case was too small to cause any symptoms, the possibility of increased growth existed. A glioma of the filum, considerably larger than my tumor, causing isolated compression of the nerves of the bladder, was reported by Lachmann²⁷. The diagnosis of vesical carcinoma was made, but the necropsy showed that the symptoms were due to paralysis of the vesical nerves and the systemic disturbance resulting from this paralysis.

As a contrast to this large glioma of the filum, Schlesinger (l. c.) reports a glioma in this location the size of a millet seed.

Although such tumors are exceedingly rare, it is well to

²⁷ Lachmann, *Archiv für Psychiatrie*, Vol. XIII, 1882, p. 50.

bear in mind that isolated paralysis of the nerves innervating the bladder may be due to a tumor of the filum. These nerves arise low in the sacral region of the cord, and, therefore, occupy a central position within the cauda equina, and are in close proximity to the filum. Enlargement of the filum may cause pressure first of all on the vesical, and possibly the genital nerves, and lead to symptoms of vesical paralysis and loss of sexual power without disturbance of motion or sensation in the lower limbs, even to the mistaken diagnosis of carcinoma of the bladder, as in Lachmann's case.

85. UEBER DEN FASERVERLAUF EINZELNER LUMBAL-UND SACRALWURZELN IM HINTERSTRANG (Concerning the Course in the Posterior Columns of Individual Lumbar and Sacral Roots). Karl Schaffer (*Monatschrift für Psychiatrie und Neurologie*, 5, 1899, Nos. 1 and 2, pp. 22, 95).

Schaffer reports three interesting cases in which he was able to study the course of individual posterior roots. In the first case the fifth lumbar root in man was degenerated; in the second the last lumbar root in a cat was completely degenerated and the upper sacral roots partially degenerated, and in the third the coccygeal and the fifth sacral roots in man were degenerated. He compares his findings with those published some years ago by C. Mayer. Schaffer believes that the descending branches of the cervical posterior roots occupy the most lateral portion, and those of the lumbar roots the most median portion of the posterior columns. The comma zones in the posterior columns, according to his views, consist of endogenous fibers, but they contain also some of the descending posterior root fibers. In his first case, the fifth anterior lumbar root was degenerated as well as the fifth posterior root, and the degeneration of the former was believed to be secondary to that of the latter. The degeneration of the roots in this first case was due to increased pressure of the cerebro-spinal fluid and to toxic and nutritive conditions produced by a tumor pressing upon the upper part of the thoracic cord. The degeneration in the second case was the result of pachymeningitis, and the case seemed to show that the intramedullary portion of the posterior roots is less resistant than the extramedullary portion. In the third case the dorso-medial sacral bundle was not degenerated, and Schaffer thinks that his case proves that the fibers in this bundle are endogenous, and that the coccygeal and sacral nerves have no part in the formation of the triangular zone. [The study of this case was not made with the Marchi method. Schaffer speaks in this paper of the superiority of the Marchi method over that of Pal, and it is certainly not necessary to remind so skilled a neuropathologist that the Marchi method often reveals a considerable degeneration where the Pal gives little indication of such alteration. The correctness of his statement: "Somit haben die Steiss und Kreuznerven mit dem dorsomedialen Sacralbündel nichts zu thun," seems unproven.]

SPILLER.

DO EPILEPTICS RECOVER?

By Edgar J. Spratling, B. Sc., M.D.,

FIRST ASST. PHYSICIAN, MASS. HOSPT. FOR EPILEPTICS; MEMBER OF THE SOCIETY FOR THE STUDY OF EPILEPSY; MEMBER OF THE AMERICAN PUB. HEALTH ASSOCIATION, ETC.

This question has so long been discussed by every one concerned in the study, treatment and family history of epileptics, without any definite conclusion being reached, that it now seems almost futile for any one even to broach the subject.

The writer believes that a very small percentage of epileptics actually do recover, but that a new onset of the disease is a fate hanging immediately over every former sufferer. Epilepsy, being a pure neurosis and having convulsions as its chief manifestation must be liable to remissions and renewals. Simply because a man has gone one month, or two months, or twelve months, or so many years, without a convulsion, should not for a moment induce us to state that epilepsy, with him, becomes in the future impossible or even improbable. So far as the writer has been able to investigate the life-history of so-called recovered epileptics, almost without exception, either that disease, or one of its numerous congeners, has fastened itself later upon the unhappy victim.

One bright young fellow, of a Hebrew family, had convulsions in infancy, grew up free from every trouble until he was twelve years of age, when by some untoward condition, from the history let us believe an enteric indigestion, pronounced epileptic seizures became frequent. Every suitable medicine, every suitable method known to medical science were employed, only leading to disappointment. At that time, after having suffered almost daily for four years, he went to Carlsbad, Germany, where either by the influence of the waters, the outdoor life, or the food, convulsions shortly ceased, and until his twenty-sixth year he was free from any evidences of epilepsy; even then epilepsy did not return, but the more fatal, yet happier paresis overtook him, with its attending convulsions, which we speak of as only *epileptiform*. Whether this fatal disease placed him beyond the reach of epilepsy, and cut short a miserable existence, or whether it blighted bright hopes and a splendid future, we are unable to say; but certain it is in this case we

have had two so-called recoveries, one in infancy, and one at sixteen.

If epilepsy, as is slowly being conceded by the most observant thinkers to-day, is simply a manifestation of some form of malnutrition, stating a prognosis is mere guess work, so long as we do not know what form this perversion takes, or what changes it works in the intimate structure of the individual cells.

For practical purposes it is the custom of those who have the care and treatment of epileptics, to place two years free of convulsions as the limit of the epileptic condition. From Craig Colony lately seven have been discharged who have measured out this apprenticeship; but whether they go out into the world on a par with their fellow-beings, whether they can enjoy life to its fullest, or whether they are to be forever handicapped and restricted by the narrow limits of a tiresome dietary, and Puritanical habits, remains yet to be seen.

This is not a disease like typhoid fever or pneumonia, in which a specific germ must be present, and must multiply its kind, and live a life-history within itself, to produce definite symptoms and certain signs; but it is a disease which determines largely its own modes and manners of making itself felt; in other words it is purely functional in its manifestation, though fundamental in its origin.

We have one patient in whom an ounce of pork will produce a series of from five to a dozen convulsions; without this pork he will go for weeks, and even months (other things being favorable) without a single untoward accident. Would we call the permanent withdrawal of pork, provided his health remained good, a recovery? Not any more than we would say that tuberculosis, which had allowed us a short period of normal respiration, was cured. We have another patient, whose father declares that a single ear of roasted green corn, was the origin of all his trouble; and even to this day, he says that green corn in any shape will produce a series of convulsions. Let us say, however, that there are other things that produce like result; as is shown by the fact that he has attacks repeatedly without eating corn. Another patient is unable to take shell-fish; and in others, alcoholic drinks will bring on a seizure within a few hours. Any number of patients may be made to have

from one to a dozen convulsions at will, by giving them an acid sauce, as cranberry, then sweet milk. The permanent withdrawal of these hurtful articles would not be recovery.

We may take another view, even into another field of epilepsy, the Jacksonian and the traumatic. How many disappointments can we count that have followed the surgeon's knife and trephine? Every one knows that even where epilepsy is caused by depressed fracture and pressure on the brain, and an operation relieves such depression, the cure at best is only temporary. Even there we see that we have not reached the fundamental perversion that allows the seizures. To say that the nervous elements have been educated, or allowed to educate themselves, into a convulsive state, is mere nonsense. No man would think and seriously assert that the nerve cell, *per se*, could explode periodically and recover its normal status of its own free will. Epilepsy is not, and never will be, considered a habit-neurosis. Therefore, the removal of mere inciting causes, as depressed fractures, or cicatrices from the cerebrum, or adherent prepuces, or troublesome nails, cannot in any sense of the word be considered as curative, but must be regarded as simply an alleviative measure. There may be rare cases in which some one single inciting cause is so powerful as to be the one determining factor in the successive seizures, and in that case, after its removal, and until another equally potent inciting cause arises, we may pat ourselves on the back, and say that we have made a cure; but we should not allow our self-esteem, or our forgetfulness of actual facts to so carry us away. We may be sure that the individual who has proven his nervous system to be so unstable as to allow repeated functional convulsions, will never pass through life unfettered, but fearful always of the dark hour that may return.

Dana, in his work on nervous diseases, says that five or ten per cent. of epileptics recover; but he fails to define recovery. Nothnagel, and numerous other writers, make the same unqualified assertion, while but few writers have candidly said that the so-called cure of epilepsy is merely the granting of the remission, not the permanent state of regained health.

The writer knows one epileptic twenty-one years of age, who had the first convulsion at seventeen, the second at eight-

een, and the third only a short time ago. He is a sufferer from spinal curvature, a weak, ill-shapen dwarf; and yet he went one year between his first and second convulsions, and two years between the second and third. Supposing he were to go two years now without an attack, should we call him recovered? A few days ago we should have done so. He states that only overloading his stomach with candy will produce a convulsion. If he should let candy alone, has a cure been wrought, or a respite granted?

The length of these remissions seems to have but little influence on the actual prognosis. If memory serves me right, one of the cases discharged from Craig Colony had had a checkered career in state asylums, county alms-houses, and was often picked up on the street as an unconscious vagrant; he had an average credit of one hundred to one hundred and fifty convulsions a month. One hundred and ten attacks were charged to him during his first month at Craig Colony, yet that man recovered, as custom speaks. Here is the lad referred to, twenty-one years old, with only three convulsions in four years, and we dare not call him recovered. The prognosis really rests upon the removability, or non-removability, of the fundamental cause, and not upon the number or severity, or any other conditions, that are apparent in the seizures.

If we find one patient suffering from too much uric acid, we cut off his meat, and limit his consumption of the pulses; and he improves, perhaps enjoying long remissions, possibly of years, yet we would not think that a cure had been wrought, so long as the uncertain fate of future seizures hung over him. Sometimes we find a man who has abused his liver with drink, his kidneys have become involved, and by physiological hygiene we relieve these abused organs, and may be rewarded for our work by long remissions.

Now the question remains, do we cure epilepsy and allow the patient to recover; or do we grant immunity, as if we were using a higher vaccination? A very few cases do actually get well, and remain so, by some structural change wrought in the cell organism; but the most recoveries are simply respites granted by the removal of immediate causes. Then let us not forget our duty to the patient, to his friends, and to the public,

and while we set a limit upon his detention, pronouncing him cured or recovered after one year, or better two years, of freedom from convulsions, at the same time let us not forget our higher duty in protecting society, by warning him against begetting offspring and forming entangling family alliances, which he would most likely be unable to carry to a happy termination.

86. RECHERCHES SUR L'ORIGINE RÉELLE DES NERFS CRANIENS. LE NERF GLOSSO-PHARYNGIEN ET LE NERF VAGUE (Researches on the Real Origin of the Cranial Nerves. The Glossopharyngeus and the Vagus). A. Van Gehuchten (Journal de Neurologie, Nos. 22, 23 and 25, 1898, and No. 2, 1899).

Van Gehuchten finds that in the rabbit the ventral nucleus of the vagus forms a long column of cells of more than four millimeters in length. This column begins just below the facial nucleus and extends as far as the lower border of the hypoglossal nucleus. The ventral nucleus on its inner and most proximal portion contains cells belonging to the ninth nerve. The ventral nucleus is sharply defined from the surrounding cells only in its uppermost part. The vagus nerve receives no fibers from the hypoglossal nucleus or from the intercalated nucleus of Staderini, and contains no crossed fibers from the nucleus on the opposite side of the medulla oblongata. Intracranial section of the eleventh nerve shows that the nerve of Willis does not arise in the ventral nucleus of the vagus, and that the dorsal nucleus gives origin to fibers belonging to the accessory nerve of Willis and the vagus. The glossopharyngeus has no origin in the dorsal nucleus. Van Gehuchten acknowledges that he was mistaken in his former opinion, and now states that the cells of the terminal nuclei of peripheral sensory nerves do not undergo chromatolysis during the first few days following section of the nerve. He has succeeded in staining the cells of the dorsal nucleus with the silver stain, and has traced the axis cylinders of these cells into the tenth nerve. He has, therefore, given us the very best proof that the posterior nucleus of the vagus is motor.

The method of Marchi shows that the sensory fibers of the tenth nerve form a large part of the solitary bundle in the rabbit. The sensory nucleus of the vagus lies between the solitary bundle and the dorsal motor nucleus of the vagus. Van Gehuchten proposes to call this the nucleus of the solitary bundle.

If the glossopharyngeus is cut at the base of the cranium, chromatolysis occurs in a small group of cells situated on the inner side of the ventral nucleus of the vagus at its upper part, and immediately below the internal column of cells of the facial nucleus. This group is not found in many sections, and represents the motor nucleus of the ninth nerve. The glossopharyngeus nerve forms part of the solitary bundle, as shown by Marchi's method, and at least the proximal portion of the nucleus of the solitary bundle is the terminal nucleus of the sensory fibers of this nerve.

SPILLER.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

March 7th, 1899.

The President, Dr. Frederick Peterson, in the chair.

SENSORI-MOTOR PALSIES OF THE MUSCULATURE OF THE FACE,

WITH REMARKS ON OCULO-MOTOR PALSIES IN THE EARLY STAGES OF TABES.

Dr. Joseph Fraenkel presented a paper with this title. He said that in tabetic patients a characteristic facial expression was often observed, and that he had given this subject special attention since presenting to the society, about one year ago, a case which had greatly impressed him with the diagnostic significance of facial expression. The case referred to was that of a musician, forty-two years of age, who had been first seen by him when presented to one of the medical societies as a case of tic douloureux. At that time the man had complained of severe pain in the area of distribution of the fifth nerve. To speak briefly, this man had presented motor impairment of the facial and ocular musculature, particularly of the left side. The defects had been chiefly those of co-ordination rather than of the motor power proper, as shown by the fact that the extent of the paralysis had varied on different occasions, and that exercise of the eye muscles had improved the motility. It was important and convenient to endeavor to establish an analogy between the cranial and spinal nerves, and between the cranial and spinal anterior and posterior roots. After a careful review of the literature Dr. Fraenkel said that he had made a careful study of 22 cases, 17 being males and 5 females. In 6 cases venereal infection was probable, in 7 there was no indication of it, and in the remainder it was admitted. Five cases without syphilitic history and eight with such history had no disturbance of the external muscles; three without, and six with it showed disorder of the functions of the external eye muscles. Seven showed marked disorder of the fifth nerve. In two the diplopia was transient. In some of the cases in which there was apparent paralysis of the external eye muscles, persistent exercise diminished this apparent paralysis. He concluded that it was possible that ocular palsies of the early stages might not be purely motor, but sensori-motor paralyses. Disease of the fifth nerve, in his opinion, had a de-

cided effect on motility, but no single function of the trigeminus could be justly accused of being the cause. The effect of disease of the fifth nerve on the motility of the eye seemed to be similar to that upon the face. Some of the ocular palsies of the early stages were probably sensori-motor.

Dr. Edward D. Fisher said that he had not specially examined most of his cases with reference to the fifth nerve. As a rule, he had not found much disturbance of this nerve, possibly because he had not carefully searched for it. With regard to the oculomotor paralyses occurring early in the disease, he believed the majority of them were transitory, and in that condition it was not difficult to imagine that the explanation given by the reader of the paper might be correct. The permanent paralyses usually occurring later, either in the form of ptosis or affecting the muscles of the eyeball itself, he was inclined to ascribe to absolute disease of the nerve itself. In the early stages, strabismus was often noticed, and the irregularity of the muscular action did not seem to be wholly due to ataxic movements of the eyeball. If the eye were at rest, there should be no strabismus, if he grasped the idea presented in the paper. Ataxic movements could not be exhibited constantly, but only when the patient changed the direction of his vision. He had not observed the condition of the facial muscles dwelt upon in the paper.

Dr. Joseph Collins said that he was glad to avail himself of the opportunity to say a word about the so-called ocular palsies of tabes. He was decidedly of the opinion that true paralyses of the ocular muscles were of such rare occurrence in this disease that they scarcely required consideration. Paralysis of one or more of the ocular muscles was not infrequently seen in the early stages of true syphilitic tabes, in contradistinction to metasyphilitic and other forms of tabes, in which if ocular palsies occurred at all they must be looked upon anatomically as evidences of nuclear degeneration in the pons, and clinically as epiphenomena, and not as an integral part of the disease. This might seem a radical and sweeping statement, yet from his experience with the disease, which was by no means very limited, he would say that with these exceptions true ocular palsy was not an ancillary manifestation of true tabes dorsalis. He had not heard Dr. Fraenkel's explanation of the diplopias, strabismic states and other forms of ocular troubles whereby the axes of the eyeballs lost their parallel in the early and later stages of this disease, but for himself such conditions were explainable on the hypothesis of a loss of reciprocal relationship between the sensory impulses and the motor responses. In other words, they were comparable to the acute ataxia which was often seen to develop in an extremity. Although the ataxia in the extremities was rarely so intense as to cause paralytic symptoms comparable to those constituting the basis of the diplopia, it should be remembered that the movements of the former were not physiologically so delicately co-ordinated and, therefore, so functionally upset by slight loss of balance. He did not deny that true genuine palsy did sometimes occur, but the explanation of these had already been offered. As to perversion of function in the domain of the fifth nerve, he believed that it was not at all a very rare condition even in those cases that had no clinical conformation to so-called high or cervical tabes. This disorder of function must be posited to explain the "tabic face," which if not so striking or characteristic as the Parkinson face or the Hutchinson face, was still very readily recognizable by one who had had much dealing with the disease. It was difficult to describe of what this facies consisted, but there was a something made up of

difference in palpebral aperture, condition of the pupils, changeable asymmetry of the face and disparate expression between the upper and lower segments of the face that was rather pathognomonic.

Dr. Fisher said that he was surprised at the statement of the last speaker regarding ocular palsies in cases of tabes. The existence of these conditions could hardly be denied; the difference of opinion should be only in regard to the explanation of them.

Dr. L. Stieglitz said that he differed very materially from the reader of the paper regarding the conception of the ocular nerve palsies. There seemed to be no necessity for ascribing the ocular nerve palsies, as seen in both the early and late stages of tabes, to ataxia. The whole clinical picture was not that of ataxia. There was an acute and absolute paralysis lasting a few weeks, and then it gradually improved, or perhaps disappeared. After recurring once or twice the picture might simulate partial ataxia. All had seen cases of genuine tabes in which there had been very distinct peripheral palsy, e. g., drop-foot. Such a case ran the course very similar to that of ocular nerve palsies, the recovery taking place as a rule within a few weeks. In tabes the motor neurons were sometimes involved without any direct connection with sensory neurons. The sensory disturbance in the first—the involvement of the fifth nerve—was not very uncommon, but occurred, as a rule, rather late.

Dr. Collins said he did not wish to draw any parallel between ataxia of the eye muscles and in the extremities, but wished to state positively that there was no such thing as ocular palsy in tabes, bearing in mind the exceptions already mentioned. In his experience, true muscular palsies in true and uncomplicated tabes did not occur save as accidental conditions.

Dr. Fisher remarked that the only contention then seemed to be in regard to what was understood by the term "tabes."

Dr. Peterson said that the paper was a valuable contribution to the clinical symptomatology of tabes, and the suggestions were worthy of the most careful study. He felt, with Dr. Stieglitz, regarding the statements made by Dr. Collins—they seemed very radical. It seemed to him that genuine ophthalmoplegias in the metasyphilitic cases were so common that they could not be considered as exceptional.

Dr. Fraenkel, in closing, said that all had seen in tabes acute ataxia in the upper and lower extremities, and had seen it disappear, with or without treatment, after two or three weeks. The explanation must be found in the progress of the disease. He would not state that the final ocular palsies of tabes had no more material basis than sensory disturbance.

REPORT OF SIXTEEN OSTEOPLASTIC RESECTIONS FOR INTRACRANIAL DISEASE.

Dr. George Emerson Brewer read this report. He said that the brilliant results early achieved by Horsley and Macewen had resulted in an amount of enthusiasm which had brought scores of sufferers to the operating table, who not only had not been benefited but had often been made worse, because wholly unsuited for such operative interference. During the past five years from thirty to forty cases of intracranial disease had fallen under his observation at the City Hospital, and nearly all of these had been seen in consultation with the neurologists

of the hospital. The object of the present report was to furnish unbiased data for further study of this important subject. Of the 16 cases coming to operation 7 had been in fair physical condition, while 9 had been recently subjected to traumatism, or were in conditions of more or less pronounced sepsis or alcoholism. Of the 7 non-septic cases 6, or 84%, recovered, the wounds all healing primarily. Of the 9 septic cases, or those in which unfavorable results were to be expected, 3 recovered. In 13 out of the 16 cases a lesion sufficient to account for the symptoms had been found. While in two instances the operation had undoubtedly saved life, both of these patients had subsequently died, one after three months from recurrence of the growth, and the other from a disease entirely independent of the condition of the brain. Five of the patients were still living. In none of the cases that had terminated fatally had there been any possibility of improvement without operation; in 5 death would have occurred almost immediately without such interference. In 2 cases death had resulted from the operation, one from acute uremia, the other presumably from an acute septic infection.

Dr. Pritchard said that he wished to place on record the history of a case that had come under his care over one year ago. The patient had had Jacksonian epilepsy along with symptoms which made the diagnosis of tumor quite evident, and which localized it quite accurately. The tumor had been found at the operation, but it had been considered inoperable because of its nature and situation. It was located in the posterior and upper Rolandic region at about the level of the junction of the leg and arm center, and rather more posteriorly than anteriorly. It was about three-fourths of an inch beneath the surface, and its superficial area was estimated to be about two inches and a half in diameter. The surgical procedure, therefore, had been nothing more than an exploration. In view of the fact that the symptoms had been growing rapidly worse before operation, and that the man had been apparently restored to health since then, he was inclined to attribute the improvement to the surgical interference. He believed it to be generally conceded that exploratory operations had often resulted in amelioration and even in disappearance of many symptoms, and his personal observations had been corroborative of this.

Dr. E. D. Fisher said that he thought the reader of the paper had shown by his results that one was absolutely justified in making exploratory operations in these cases. It had been stated that only two of the cases had died as a result of the operation, and in one of these he felt sure that it was not fair to attribute the death to the operation, but in the absence of an autopsy-record this had been thought the most warrantable assumption. The paper especially emphasized the comparatively little danger of these exploratory operations in competent hands.

Dr. Collins said that he thought the value of the contribution of Dr. Brewer lay chiefly in teaching us that intracranial surgery had a very limited field as a therapeutic agency. Until this view had been generally accepted, he thought the indiscriminate intracranial operations, so common among surgeons at the present day, would continue. At the

last meeting of the British Medical Association Ferrier had presented the results of his work. He claimed to have benefited 35 per cent. of his cases, and 17 per cent. of these had been absolutely cured. Of the remainder he claimed one to five years had been added to their lives. Dr. Collins said that in the discussion on that occasion he had expressed his surprise at these results, in view of the fact that the usual percentage of recoveries was thought to be about 1 per cent. to 5 per cent. In a recent letter received from Ferrier the same position had been maintained, and the explanation offered was that he had selected his cases for operation with greater care and more diagnostic discernment than he had expended on any other class of cerebral disease. This must be the keynote to all future intracranial surgery. With all due respect to the skill of Dr. Brewer, he felt that the cases should have been selected with greater care, for, in at least two or three of them it seemed to be a foregone conclusion that the operation could not be beneficial. The statement had been repeatedly made in the paper that the dangers of the operation had been explained to the patient and had been accepted, but it should be remembered that these patients were not in a condition to judge. He mentioned this to warn surgeons against allowing themselves to be influenced by the statements or wishes of these patients. He believed intracranial surgery should be limited to sinus thrombosis, intracranial abscess and a very few cases of intracranial tumor.

Dr. Stieglitz said that this candid report was of great value, although he agreed with the last speaker that the field for such work was very small. The pyogenic cases offered a far better field than did epileptic cases or intracranial tumors. The experience of Macewen in the first class should lead the surgeon to undertake operation, even in apparently severe cases. The cases of intracranial tumor which had been successfully operated upon were practically those of new growths in the motor area, although occasionally success might be achieved in operations on tumors of the cerebellum.

Dr. Peterson remarked that as much was to be learned from both successes and failures, the paper was really a valuable contribution.

Dr. Brewer, in closing the discussion, said that under ordinary circumstances these brain operations could be undertaken in hospital practice without danger of sepsis. In only one of these cases, which was not septic at the time of operation, did sepsis occur, and he was willing to admit that this should have been avoided. None of the epileptics were apparently made worse by the operation, but, of course, if local infection occurred, the operation was liable to greatly aggravate the condition. It was quite probable that in nineteen-twentieths of the cases of epilepsy operated upon the operations were fruitless, still it was well to undertake the operation in recent cases in the hope that occasionally a person would be saved from a life of epilepsy. He had always felt that even in beginning septic meningitis there was a possibility of affording relief by operation. He proposed as soon as opportunity offered to try the effect of irrigation in a case of this kind.

PHILADELPHIA NEUROLOGICAL SOCIETY.

February 27, 1899.

Vice-President, Dr. John K. Mitchell, in the chair.

A CASE OF ATAXIA FOR NOSOLOGICAL DIAGNOSIS.

PROBABLY PROGRESSIVE MENINGO-MYELITIS (CEREBRO-SPINAL).

Dr. F. Savary Pearce presented a case of ataxia for diagnosis. A sister of the patient became deaf in her eighth year, apparently from central cause. The father was alcoholic. The family history otherwise was negative.

The patient, a man of 22 years, began to be deaf at the age of eight, like the sister, from unknown cause. This deafness became almost absolute in a few years, and has remained so. No history of venereal infection was obtained. The present trouble began more than three years before he was first seen by Dr. Pearce, on May 1st, 1898. In 1895 he first complained of rather sudden failure of vision, vertigo, tinnitus aurium, frontal headache and gastric indigestion. In November of that year the pupils were 2 mm. in diameter, and irresponsive to light and in accommodation. Retinal detachment, with swelling of the optic disk, was also found in the right eye, and vision was reduced to light perception. The optic disk was also swollen in the left eye, and vision was $\frac{5}{125}$ (?). The field was slightly contracted for form and colors. The treatment consisted of Donovan's solution, potassium iodide and special movements. In November of 1898 the pupils were pin-point in size and fixed; marked rotary nystagmus existed in both eyes; the deafness was constant; tinnitus aurium and muscæ volitantes were complained of; the vision was $\frac{5}{125}$ (?) in O. S.; the gait was very ataxic, as it had been for over two years, and the patient swayed much when standing erect, even with the eyes open. He complained of formication in all the extremities. Sensation throughout the body was markedly obtunded, and a small saddle-shaped area of hypæsthesia existed in the perineum, but no anesthesia was found anywhere. A trophic ulcer was on the left buttock. The wasting of the muscles was general. No tenderness over the nerve trunks existed. Incontinence of urine was noted at times, and the bowels were costive. The knee-jerks were absent at first, and in February of 1899 could be obtained by reinforcement, but were soon exhausted. Both optic nerves were in a condition of advanced atrophy.

Dr. Charles K. Mills thought that the possibility of the case being a form of cerebro-spinal syphilis had not been ruled out. He also sug-

gested the possibility of it being a case of gliosis, affecting both the cord and the basal region of the brain, in the peculiar manner in which this process does sometimes attack these regions.' He thought that a cerebellar lesion would not explain the whole case.

Dr. Francis X. Dercum said that some of the symptoms certainly suggested cerebellar lesion; e. g., the ataxia, the titubation, the nystagmus, the variability of some of the symptoms; as did also the fact that the knee-jerk was elicited with difficulty at one time and more readily at another time. He asked whether it were not possible that we had here syphilitic disease of the cerebellum and also of other portions of the nervous system, whether it might not be hereditary syphilis. Hereditary syphilis not very rarely produces symptoms of organic nervous disease in the adult. The case suggested not only diffuse nervous syphilis, but also gumma of the cerebellum. Gumma in hereditary syphilis is rare, but cases have been reported. The fact that improvement followed the use of Donovan's solution and the iodides also indicated specific disease.

UNILATERAL ARGYLL-ROBERTSON PUPIL.

Dr. Wendell Reber said that in 1894, in an article on unilateral reflex iridoplegia, or unilateral Argyll-Robertson pupil, Haddaeus¹ (of Essen, Germany) made the statement that up to that time there had been reported to his knowledge but five instances of uncomplicated unilateral Argyll-Robertson phenomenon. This statement, along with that of Mr. Bevan Lewis, who found the anomaly but 11 times in 147 general paretics; Dr. Berger, who found it 3 times in 109 tabetics, and Dr. Gowers, who found it once in 72 tabetics, would indicate sufficient rarity to justify the report of the two following cases, for the observation of which he was indebted to the courtesy of Dr. William Campbell Posey:

CASE I. R. W. P., male, married, *æt.* 60, an inmate of the State Hospital for the Insane at Norristown, Pa., was admitted to that institution August 29, 1898, in a condition of chronic dementia. Physical condition good. Family history not obtainable. Urinalysis: sp. gr. 1032, reaction acid, no albumin or sugar, centrifugalized sediment showed only occasional urates and bladder epithelium. The patient's gait and station were fair, but there was abolition of the knee-jerks. Symptoms in the visual sphere. Tremor of the eyelids when the eyes were gently closed. Vision equaled 5-10 in both eyes, and with + 4.00 spherical the 0.50 D type was read from 30 to 40 cm. The ophthalmoscope showed in the right eye a disk of fair tint, oval in its vertical diameter, bounded all round by a visible scleral ring and skirted to the outer side by a well marked choroidal crescent. The vascularity of the fundus was good, likewise the condition of the outlying choroid and retina. The conditions in the background of the left eye were practically the same as those of its fellow. The pupillary status was as follows:

R. E. pupil $3\frac{1}{2} \times 4\frac{1}{2}$ mm.; long axis 90°. Did not react to direct or consensual light stimulus, but reacted fully $2\frac{1}{2}$ mm. to convergence stimulus.

L. E. pupil 4×5 mm.; long axis 90°. Responded 1 mm. to direct

¹ Knapp's Archives, Vol. xxiii.

and $\frac{1}{2}$ mm. to consensual light stimulus, and fully $2\frac{1}{2}$ mm. to convergence stimulus. Therefore, while this eye, strictly speaking, could not be said to exhibit the Argyll-Robertson phenomenon, yet the paresis of the iris on this side portended early loss of all response to light stimulus.

CASE II. G. M. W., *et.* 29, single, also an inmate of the State Hospital for the Insane at Norristown, where he had resided for some years in a condition of epileptic dementia. The patient was rather poorly nourished, and for the last five years has averaged about ten convulsions each month. The only family history obtainable was that some of his maternal ancestors were epileptic.

Ocular signs were as follows:

Vision—R. E., 5-10; L. E., 5-7 $\frac{1}{2}$. Accommodated up to 18 cm. in both eyes. The ophthalmoscope showed in the right eye a round disk rather gray in its deeper layers, shallowly excavated throughout its extent and bounded all round by a somewhat sharp scleral ring. There was an absorbing conus to the temporal side, the arteries were narrower than normal and the veins broader. In the left eye the nervehead was oval, 6×7 diam., with its long axis at 75, and of the same hue as the right nervehead. The scleral ring was visible only to the inner and outer sides, and there was a small pigment deposit to the temporal side of the disk. The pupils were as follows:

R. E. pupil 3 mm.; did not respond to direct or consensual light stimulus or to convergence stimulus. L. E. pupil $3\frac{1}{2}$ mm.; did not react directly or consensually to light, but reacted 1 mm. to convergence stimulus. Hence, absolute iris paralysis on the right side, and reflex iris paralysis (Argyll-Robertson pupil) on the left side. A point of no little importance in this case was that the pupils were of the same diameter in the dark room as when exposed to diffuse daylight, indicating, perhaps, some implication of the sympathetic fibers to the iris. Unfortunately the cutaneous tests were not made.

In a rather hurried search through the literature Dr. Reber had found the report of 53 instances of this anomaly, and he believed that with more time at his command he could have brought more cases to light.

He said that to attempt an explanation of unilateral Argyll-Robertson pupil was to open the whole question of the path of the pupillary reflex, which has a literature as vast as it is vague. Among the latest contributions to our knowledge of this reflex is that of Bernheimer². His work was conducted on anthropoid apes and on human embryos, and combines the methods of Marchi and Golgi. He states that the pupillary fibers undergo a partial decussation in the chiasm along with the visual fibers, so that each sphincter nucleus is in relation with the visual and pupillary fibers of both eyes; that, in addition to this double connection, there must also exist a central connection between the two nuclei, not yet well established, but in all probability effected by contact of the long ganglion cell processes which, in Golgi preparations, can be seen arborizing across the median line.

Even if this hypothesis of Bernheimer be correct, it brings

² Graefe's Archiv für Ophthalmologie, Nov., 1898.

us very little nearer to the solution of this question of unilateral Argyll-Robertson pupil. Granting that the pupillary fibers of the optic nerve do decussate along with, and in exactly the same manner as, the visual fibers, and granting farther that there does exist central connection between the two sphincter nuclei, we are driven, nevertheless, to the conclusion that the oculomotor nucleus or the motor tract must be the affected region. The only region in the motor tract in which a lesion producing one-sided Argyll-Robertson pupil could occur without causing coincident palsy of some of the extraocular muscles of the same eye is the ciliary ganglion, and thus far the hypotheses having reference to this ganglion do not explain the Argyll-Robertson phenomenon nearly as well as does the nuclear hypothesis. It would seem that in the present state of our knowledge, the most we can say in explanation of either the unilateral or bilateral Argyll-Robertson pupil is that it is the peripheral manifestation of a disturbance in the most anterior portion of the oculomotor nucleus; and that this nuclear disturbance is most likely either of the nature of an obliterating endarteritis or a slow sclerosis.

Dr. G. E. de Schweinitz said that his personal experience with unilateral reflex iridoplegia was limited to two or three cases, the most marked one being a patient with cerebro-spinal syphilis in the wards of the Philadelphia Hospital, which he had studied in conjunction with Dr. Dercum. With reference to the lesion in cases of this character, he thought that all modern investigations tended to show that it must be situated in the centrifugal portion of the reflex mechanism, and was probably nuclear in origin. He referred to the admirable paper on unilateral loss of the pupillary light reflex by Dr. Leszynsky, published in the *New York Medical Journal*, August 6, 1898, in which there is a complete review of the literature on the subject.

Dr. Francis X. Dercum said that he had seen, in addition to the cases mentioned by Dr. de Schweinitz, another case of unilateral Argyll-Robertson pupil occurring in a paretic. He thought that the condition occurring in paretics was open to the objection that perhaps in a later stage of the disease the other pupil would also become involved; that there is really in such cases bilateral involvement of the pupil with the condition merely pronounced in one eye and slightly or doubtfully present in the other, the difference disappearing as the cases progress.

Dr. W. G. Spiller said he had hoped that the fibers controlling the iritic reflex would have received more attention. He mentioned that V. Bechterew had reported a case in which voluntary control over the movements of the iris existed. This seemed to show that there might be, in some people at least, a cortical center for the iris.

Dr. Spiller referred to an interesting paper published by Massant. This investigator removed the iris and obtained degeneration of the "pupillary fibers" in the optic nerve, although he did not injure the optic nerve directly, or the oculomotor centers. He removed the iris and injured the motor nerves contained within it, and following that injury he found degeneration of the "pupillary fibers" in the optic nerve; i. e., he obtained degeneration of the sensory part of the reflex arc following injury of the motor part. Dr. Spiller thought that if these observations were confirmed they would be most important. What is

true of one part of the body may be true of other parts. We may believe that in the reflex arc for the knee-jerk, fibers exist which subserve the transmission of afferent impulses and have no connection with ordinary sensation. If this is true, the knee-jerk might be lost on account of some lesion of the sensory part of the reflex arc, without disturbance of sensation; and, furthermore, from Massaut's findings, we have a right to expect degeneration of the sensory part of the reflex arc from injury of the motor part. Dr. Spiller was not aware that Massaut's results had been confirmed.

Dr. F. Savary Pearce asked of the ophthalmologists present why it was that in many cases where there was Argyll-Robertson pupil the pupils were contracted.

Dr. G. E. de Schweinitz, in reply, said that the Argyll-Robertson phenomenon might be associated either with myosis or with mydriasis. This depends upon the relation of the lesion to the pupil-contracting center or fibers, or to the pupil-dilating center or fibers; for example, the common paralytic myosis, often known as spinal myosis, which occurs in lesions of the cord above the dorsal vertebræ and is noteworthy in *tabes dorsalis*, is caused by a paralysis of the pupil-dilating fibers, and may later exhibit the Argyll-Robertson phenomenon. Again, there may be irritation mydriasis with Argyll-Robertson phenomenon, appearing because the pupil-dilating fibers in the posterior columns are irritated.

Dr. John K. Mitchell referred to a patient who had been sent to the Orthopedic Hospital by an ophthalmologist, with a diagnosis of *tabes*, from the fact that Argyll-Robertson pupil was present. Absolutely no other symptoms of *tabes* could be found, and the man is apparently still in perfect health, three months after the condition of the pupil was first observed.

A CASE OF CEREBELLAR TUMOR.

Dr. William M. Sweet and Dr. William G. Spiller reported a case of cerebellar tumor occurring in a previously healthy, well nourished girl, 12 years of age, whose family history was good. From the history given by the family, she was taken sick at school early in September, 1898, and suffered from a dull pain in the head, associated with vomiting. The pain in the head, which was a prominent symptom throughout the illness, was always frontal, never sharp in character, and not worse at night. Early in October the mental faculties showed impairment, the child taking very little interest in her surroundings; there was beginning loss of co-ordinating power in the lower extremities, and vision began to be impaired. About the middle of December the hearing commenced to fail, and frequent attacks of pain in the ears occurred, especially severe on the right side. At no time was vertigo complained of, nor was twitching of the muscles of the face or corners of the mouth noticed. Both bladder and bowels were normal in their functions.

At the time of the first examination of the child, early in January, there was marked stupor, and it was with great difficulty that she could be aroused. The loss of co-ordinating power of the lower extremities was almost complete, and when

trying to walk the tendency was apparently to fall backward. The knee-jerks were entirely abolished, although reinforcement could not be employed. No spasticity of the limbs was seen. The cranial nerves, except the auditory and the optic, were not involved. Slight involuntary movements of the fingers were detected. Sensation was felt in the hands and fingers. The speech was almost unintelligible, and deglutition was difficult.

The ocular examination showed no deviating tendencies or restrictions of normal movements of the eyeballs; corneal sensibility was unimpaired; the vision was reduced to faint light perception; the pupils were 6 mm. in diameter, round and fixed; the media were clear, but a marked papillo-retinitis was present in each eye, the swelling of the nerve head in the right side being 6 diopters and of the left side 5 diopters. In the macular region of each eye the stellate figure often associated with retinal inflammation and edema was present.

A round-cell sarcoma was found in the middle lobe of the cerebellum. It greatly compressed the surrounding tissue and completely filled the fourth ventricle. The aqueduct of Sylvius and the third and lateral ventricles were much dilated. Considerable degeneration of one anterior pyramid was found by the Marchi method, and this was evidently due to pressure, although the tumor was separated from the pyramid by a large amount of normal tissue. The degeneration of the motor tract in cerebellar tumor from pressure at distance was regarded as an interesting finding, and indicated that in cases of cerebellar neoplasm motor symptoms may be present and not result from direct involvement of the pyramidal tract by the growth. The posterior columns, especially the columns of Burdach, and the inferior cerebellar peduncles, contained many degenerated fibers by the Marchi stain. The pressure on the nucleus of the sixth nerve and the knee of the facial nerve on each side must have been considerable, as seen in macroscopical preparations, and yet no disturbance of the function of these nerves was observed during life. The gradual development of the pressure was probably the explanation of the preservation of the function.

Attention was early directed by Dr. James Taylor to the close relation existing between cerebellar tumor and the presence of the stellate figure in the macular region. It is possible, however, to have papillo-retinitis associated with cerebellar disease without the stellate figure being present, and also to have a neoplasm in the frontal lobes, with the stellate degeneration of the macular region existing.

Whatever may be the cause of this stellate figure, clinical observation associates it with retinal inflammation and edema. It is not alone seen in brain tumors and in renal disease, but

has been found in anemia, in thrombosis of the central retinal artery, in typhoid fever, and similar affections, with which papillo-retinitis is sometimes an associated condition. Marcus Gunn believes that the condition is due to the retinal effusion being arrested at the fovea by the close attachment of the structures at this point, this pegging down of the swelling tissue causing a folding of the retina radiating from the fovea. These fine radiating folds favor degenerative processes and tend to assist in the collection and coagulation of inflammatory exudate.

Dr. G. E. de Schweinitz said he had seen the star-shaped figure in the macula in cases of cerebellar tumor, in one case of tumor situated in the motor region of the brain which it was impossible to remove by a trephining operation, and in other cases of optic neuritis which were unassociated with intracranial lesions. The two most noteworthy of these were unilateral, one in a girl between twenty and thirty, which had arisen without evident cause, and which, in the course of a couple of years, entirely disappeared, and the other in a medical man following an attack of typhoid fever. Dr. de Schweinitz thought that Mr. Gunn's explanation of these cases, already defined by Dr. Sweet, was satisfactory, but he was quite sure that it would be a mistake to suppose, as at one time had been suggested, that the star-shaped figure in connection with choked disk was diagnostic of cerebellar tumor.

Dr. Spiller agreed with the statement made by Dr. de Schweinitz, and referred to a case shown him by Dr. Thomson, a little over two years ago, at the Orthopedic Hospital. Areas resembling those of albuminuric retinitis were seen about the disk, and were such as are regarded as characteristic of cerebellar tumor. Dr. Burr found a tumor of the frontal lobe and none in the cerebellum in the post-mortem examination of this patient.

REVERSAL OF THE PERCEPTION LIMITS.

Dr. M. W. Zimmerman, by invitation, presented the visual fields and clinical histories of thirteen cases which exhibited the phenomenon of partial reversal of the perception limits. Six of these cases were undoubtedly hysterical; five, in addition to grave organic lesions of toxic origin, justified the suspicion of mild hysteria as a secondary result, and two were instances of acute local ocular lesions with no hysterical symptoms whatever. He valued the visual field changes in the diagnosis of hysteria as follows:

1. Moderate contraction of the visual field *alone* is not conclusive.
2. Extreme contraction in which the field for white or form suffers relatively more than the color fields, suggests very strongly the presence of hysteria.
3. Complete reversal, and persistent reversal, when of considerable extent and not explained by organic lesions, are almost certain evidences of the hysteric state.

Dr. G. E. de Schweinitz agreed with the essayist that the perimeter conscientiously and carefully used yielded accurate results. The

relation of the color lines to each other in perimetric examination was interesting, and reversal might be expected as a natural phenomenon when the eyes are examined in a slightly darkened room, to the light of which the retina has not yet become adapted; in cases of hysteria and allied conditions; in certain ocular diseases, especially when these are associated with marked depreciation of general nutrition, notably xerophthalmos, as has been particularly pointed out by Mr. Stephenson; in toxemias, for example, nitrobenzol poisoning, chronic lead poisoning, etc., and in certain organic ocular diseases—optic nerve atrophy, retinitis, etc. In conjunction with Dr. John K. Mitchell, Dr. de Schweinitz had studied many cases of hysteria with reference to the visual field, the first paper having been published ten years ago. They had failed to find the marked achromatopsia described by the French observers in hysterical cases, although they had often noticed reversal in the normal sequence of the colors, so that red was usually the largest field. This was generally present when there was anesthesia, but disturbance of the color-sense and anesthesia did not necessarily belong to each other, because they had examined at least two cases of universal anesthesia without finding alterations in the visual field, and a third case in which, although there was marked contraction, reversal was not present. They had several times noted complete reversal in the color lines, one of the most remarkable being a patient with hemiplegia which had been studied in several hospitals and had sometimes been regarded as an organic hemiplegia, and at other times as an hysterical paralysis with anesthesia.

Dr. William G. Spiller asked whether any of the ophthalmologists present had met with cases of hysterical hemianopsia. He had seen such a case in Dejerine's clinic, and believed that this was the first on record. He had also seen a second case presented by Janet at one of Raymond's lectures.

Dr. G. E. de Schweinitz said that the condition was exceedingly rare. One case had been reported by Dr. John K. Mitchell and himself. Dr. Lloyd, he thought, had reported another.

He had once taken the fields of vision of a hysterical woman while she was in a semi-hypnotic condition, but had not found an anomalous arrangement of the color lines.

87. HYPERTROPHIE UNILATÉRALE DU SEIN DANS L'HEMIATHETOSE INFANTILE (Unilateral Hypertrophy of the Breast in Infantile Hemiathetosis). Lannois (Lyon Medical, 88, 1898, p. 510).

When infantile cerebral paralysis influences the nutrition of the breast it is ordinarily to cause atrophy (aplasia?) but hypertrophy is not unknown, and the author reports two examples. The first was in the person of a young woman, aged 19 years. The patient had had a cerebral hemiplegia with marked athetosis on the affected side and severe epileptic seizures since her second year. The left (affected) arm was considerably hypertrophied, and the left breast exceeded the right by five centimeters in circumference, and by 1.8 centimeters in the diameter of the areola.

The second patient was also 19 years old, the subject of infantile cerebral (left) hemiplegia and athetosis, but not epileptic. Like the first, this patient also showed some hypertrophy of the left arm, and the left breast measured 3.5 centimeters more in circumference than did the right.

PATRICK.

Periscope.

ANATOMY AND PHYSIOLOGY.

88. LE CENTRE DE L'AGRAPHIE ET LA SURDI-MUTITE (The Center of Agraphia and Deaf-Mutism). E. Brissaud (*La Presse médicale*, 15 Janvier, 1898, p. 25).

The author criticises the theory of Marie, that spoken language is the result of the activity of a preformed center, while written language comes from a center developed by the acquirement of writing. This theory does not, apparently, coincide with all the facts, particularly the facility with which children acquire foreign languages, and in particular, the ability to communicate their thoughts by means of their fingers. Brissaud thinks language is largely a matter of mnemonic reflex; that is to say, the infant perceives an object, hears its name mentioned, learns to articulate that name, and remembers it. It is equally easy, however, for it to learn to imitate a gesture, serving instead of the word, as the name of the object. He cites the interesting case of a child, well formed and intelligent, who, at the age of seven years, became totally deaf as a result of double otitis media. As soon as the deafness was complete the child became dumb. Later it was taught the finger language, which it acquired with extraordinary rapidity, and subsequently again learned phonation; that is to say, the same patient exhibited a power of functional adaptation upon three occasions, apparently independent of any preformed center. SAILER.

89. DER CENTRALE URSPRUNG DES N. VAGUS (The Central Origin of the Vagus Nerve). E. Bunzl-Federn (*Monatsschrift für Psychiatrie und Neurologie*, 5, 1899, p. 1).

Bunzl-Federn has cut the vagus nerve in rabbits in different parts of its course, and studied the chromatolysis resulting in the cells of origin of this nerve.

When the vagus was cut above the exit of the laryngeus superior, but below the exit of the ramus auricularis, most of the cells of the dorsal nucleus on the operated side degenerated, and almost as many degenerated when the vagus was cut below the exit of the laryngeus superior; showing that fibers of the latter branch are connected with few cells in the dorsal nucleus. This was shown also when the laryngeus superior was cut alone. A few degenerated cells in the opposite dorsal nucleus were found after division of the vagus.

The dorsal nucleus, even in its proximal portion, does not belong to the glossopharyngeus, because after division of the vagus only degenerated cells were found in the proximal portion of the dorsal nucleus.

When the vagus was cut below the exit of the laryngeus superior and the ramus auricularis, normal cells were found in the dorsal nucleus; and degenerated cells were found in this nucleus when the laryngeus superior was cut alone. The laryngeus superior is, therefore, certainly, and the ramus auricularis is probably, in relation with the dorsal nucleus. This is contrary to the views of Dees, who believes that the dorsal nucleus is motor. Bunzl-Federn thinks that the vagus ter-

minates in the dorsal nucleus of the same side, and is in relation with the dorsal nucleus of the opposite side.

When the vagus was cut above or below the exit of the laryngeus superior, most of the cells of the ventral nucleus (nucleus ambiguus) degenerated. Only in one out of five cases were a few degenerated cells found in the nucleus ambiguus of the opposite side. The vagus is not connected, therefore, with the contralateral nucleus ambiguus. The ventral nucleus gives origin to the vagus, but contains cells belonging to other nerve fibers, though not to the accessorius.

Cells near the central canal on the medial border of the anterior horn in the upper part of the cervical cord, and lower part of the medulla oblongata are in especial relation with the laryngeus superior.

The accessorius arises from a nucleus in the anterior horn of the cervical cord and the continuation of this nucleus in the medulla oblongata—which is not a part, however, of the vagus nucleus. The bulbar fibers pass into the vagus. The three or four more superiorly situated roots in the medulla oblongata, having their exit at the sulc. post. lat., are in connection with the dorsal nucleus of the vagus, and are united with the accessorius in a part of their course, but really belong to the vagus.

The motor fibers of the laryngeus superior, and the motor fibers of the thoracic and abdominal vagus, arise in the "dense formation" (proximal portion) of the ventral nucleus; the motor fibers of the laryngeus inferior arise in the "loose formation" (distal portion) of this nucleus.

SPILLER.

90. LA THÉORIE DES RÉFLEXES (The Theory of Reflexes). E. de Mas-sary (La Presse médicale, 3 Février, 1898, p. 69).

Two arcs are concerned in the production of reflexes; the central, composed of fibers passing from the nuclei of Goll and Burdach to the cortex, with perhaps accessory arcs through the cerebellum; and of the fibers passing from the cortex to the ganglion cells of the anterior cornua of the cord. The peripheral neurons are the accessory fibers passing through the spinal ganglia to the nuclei of Goll and Burdach, and the motor fibers from the ganglia of the anterior cornua to the muscles. Any interference with the peripheral arc produces abolition of reflex. Any interference with the central arc causes exaggeration of reflex. This exaggeration is the result of the inhibitory action of the cells of the cerebral cortex, and is, therefore, produced chiefly by a lesion placed between these and the peripheral arc, that is to say, in the pyramidal tract.

SAILER.

91. ON THE BILATERAL ACTION OF THE LATISSIMUS DORSI IN HEMI-PLÉGIA. C. E. Beevor (British Med. Journal, No. 1970, p. 976).

While examining a patient's chest the author observed that in coughing the latissimus dorsi contracted on both sides, giving a powerful expiratory effort, and this action can readily be felt by any one placing the finger and thumb on the muscle at the posterior border of the axilla and giving a strong cough. In the text books on anatomy this muscle has always been described as a muscle of inspiration, and never as one of expiration.

On placing the hand on the posterior axillary fold and inspiring deeply, some contraction can be felt, but it is very much less than the powerful movement felt on the expiration produced in coughing. It is difficult to understand how the same muscle can be both inspiratory

and expiratory, and in a previous paper the author has made the statement that there is no example of a muscle acting diametrically opposite to its usual direction of action, but the latissimus dorsi seems to be an exception. The latissimus dorsi has an extensive origin, and it is suggested that in the action of expiration the part of the muscle which arises from the iliac crest and the spine compresses the abdominal cavity and assists the expiratory movement, while in inspiration the action is produced by the part of the muscle which arises from the three or four lower ribs, elevating them. This respiratory movement is the bilateral action of the muscle, and always affects the muscles of both sides in health.

The unilateral action of the muscle is that on the upper limb, and it is best brought about by making a patient adduct to the side of the chest, the upper limb placed in the horizontal position, and in this movement the muscle acts quite independently of its fellow of the other side.

As a cough can be voluntary or reflex, we have three conditions of action of the muscle, but in the case of a sneeze, which is an involuntary expiration, only two. We have, therefore:

1. Reflex coughing or sneezing; bilateral action.
2. Voluntary coughing; bilateral.
3. Voluntary adduction of humerus; unilateral.

The next question was to examine as to the presence or absence of these actions in hemiplegia. Examination of a dozen cases revealed in all but two the following conditions:

1. In reflex coughing the expiratory action of the latissimus dorsi was about equal on the two sides.
2. In voluntary coughing the action of the latissimus dorsi was obtained on the paralyzed side, but frequently diminished in action or occurred later than on the normal side.
3. In all cases the unilateral voluntary action of adducting the shoulder-joint was absent.

PATRICK.

PATHOLOGY.

92. THE MICRO-ORGANISM OF SIMPLE POSTERIOR BASIC MENINGITIS IN INFANTS. Geo. F. Still (British Med. Journal, 1898, No. 1972, p. 1157).

The simple posterior basic meningitis of infants—a non-tuberculous form of meningitis which occurs mostly within the first year of life, and affects especially the posterior portion of the base of the brain and also the spinal cord—is a condition of no great rarity. In the earlier half of the year it is always more or less prevalent in London, and there can be little doubt that many cases which appear to be tuberculous meningitis are in reality cases of simple posterior basic meningitis. It has been shown on clinical grounds that there is good reason for believing that this form of meningitis is a specific disease, and the author's observations from the pathological and bacteriological standpoint seem to confirm this view.

The cases may be divided into three groups: (i) Those fatal within six weeks—that is, during the acute or inflammatory stage; (ii) those fatal at the end of three or four months—that is, during the chronic or hydrocephalic stage; (iii) those in which recovery occurs.

Corresponding with these variations in the duration of the disease, there are differences of pathological appearance in groups (i) and (ii). In the former there is much lymph over the base of the brain and on the spinal cord; in the latter there may be no trace whatever of lymph, only thickening and opacity of the pia-arachnoid, with ad-

hesions especially between the medulla and cerebellum. These are briefly the morbid changes found, and it is important that it should be realized that there are such differences in the morbid anatomy of this disease in its early and in its late stages. In the early stage the condition has not been sufficiently differentiated from what one might call "secondary" suppurative meningitis, particularly the common form due to the pneumococcus. In the late stage it has almost certainly been labeled sometimes as healed tuberculous meningitis, sometimes as a chronic syphilitic change.

In the simple posterior basic meningitis of infants it is almost invariably the case that no lesion, except such accidental complications as occur in any prolonged disease during the last few days or hours of life, is found in the viscera; whereas in other forms of meningitis exudation in the meninges is almost invariably secondary to some obvious focus of infection elsewhere. In fifteen consecutive cases of suppurative meningitis which were examined by the author, all except one showed an obvious primary focus of infection, while in fifteen consecutive cases of posterior basic meningitis not a single one showed inflammatory lesions elsewhere, except two, in which a lymph exudation was present about certain tendon sheaths. This fact, together with the constancy of its clinical and pathological features, seems to point to its being a specific disease due to some micro-organism. In seven out of a series of eight cases reported at the Pathological Society of London last year the author found a diplococcus, which, so far as the evidence went, appeared to be the specific cause of the disease. The one case which proved sterile died three and a half months after the onset of the disease.

Since then he has made four post-mortem examinations of infants with this disease, and in two of these the micro-organism was present in the exudation at the base of the brain, apparently in pure growth, but owing probably to the use of strong carbolic acid for the sterilization of instruments no cultures were obtained; the other two cases died respectively on the seventy-fifth and eighty-ninth day after the onset of the disease, when all exudation had already disappeared.

In another case he has recently obtained pure cultures of the micro-organism from the cerebro-spinal fluid taken from the lateral ventricles during life, in the course of an operation for the establishment of artificial drainage. It would seem, therefore that the micro-organism is present during the earlier stage of the disease, but disappears later.

For the peculiar features of this micro-organism the original must be consulted.

PATRICK.

93. MÉNINGITE CÉRÉBRO-SPINALE ÉPIDÉMIQUE (Epidemic Cerebro Spinal Meningitis). D. Assimis (La Presse médicale, May 28, 1898, p. 289).

In 1897, during the month of November, a few sporadic cases of this disease were observed. During the months of November and January these increased until an actual epidemic was in progress. The author observed altogether 16 cases, with 10 recoveries and 6 deaths. Of the latter, two cases came to autopsy. Of the 16 cases, 5 had the comatose form of the disease. In all cases, albumin was present in the urine, disappearing with recovery. Five cases had the foudroyant form. The symptoms commenced with chills, high fever reaching 41° C., intense headache, delirium, vomiting and general spasms of the whole body. In one case the body was covered with petechiæ varying in size from a franc to a 50-centime piece. Three cases had an abortive form of the disease, commencing with fever, headache and

vomiting—all symptoms disappearing in the course of two or three days. In the two cases upon which autopsy was performed, fibrinous exudates were found upon the pia mater, and serous exudate in the pericardium. A few interesting facts were brought out by the bacteriological investigations. A spheroidal coccus was obtained, sometimes isolated, sometimes in pairs, always extra-cellular, and not taking Gram's stain. This grew both aerobically and anaerobically, was found not only in the exudate, but in blood from the heart and finger, and in the tissue of the spleen. It was non-pathogenic for rabbits or white mice. Growths were obtained upon potato and bouillon, thus differing from the meningococcus of Weichselbaum. No case of direct contagion was observed. One of the nurses in the hospital, however, contracted the disease. The most benign cases were found among young infants.

SAILER.

94. ZUR FRAGE DER VERÄNDERUNGEN IN DEN PERIPHERISCHEN NERVEN BEI DER CHRONISCHEN ERKRANKUNG DER GEFÄSSE DER EXTREMITÄTEN (Concerning the Changes in the Peripheral Nerves in Chronic Disease of the Vessels of the Extremities). Michael Lapinsky (Deutsche Zeitschrift für Nervenheilkunde, 13, 1898, p. 468).

Lapinsky has examined eight cases in which the arteries of one or both lower extremities were diseased, and gangrene of the limbs followed. The clinical evidences of neuritis were absent; motion and sensation were about normal. The vasa nervorum were altered, and the connective tissue of the nerves was much increased in amount, especially toward the foot, and this increase was proportionate to the change in the vessels. The nerves contained numerous spaces, supposed to be due to stagnation of the lymph. The nerve fibers were not greatly altered, except that where the endoneurium was much thickened the nerve fibers were small. These changes of the nerves were regarded as the result of vascular disease.

SPILLER.

95. NERVOUS VOMITING. Bertram Hunt (Clinical Journal, Vol. XII, 1898, p. 239).

The author reports what he believes to be a fatal case of functional emesis. A married woman of 29 years had had, a year before, an attack of abdominal pain and vomiting which lasted several days and then left her entirely. Nine months after this she had a severe "bilious attack," and on the following day vomited a teacupful of bright blood. From this day to the time of admission she constantly vomited directly after meals, and also between meals, there being occasionally a small quantity of blood in the vomit. When admitted the patient was but slightly emaciated; the abdomen was rigid and tender all over, but she referred the spontaneous pain entirely to one point in the epigastrium, pointing to the painful spot with one finger in the way described in the text-books as typical of gastric ulcer. The treatment consisted of cutting off all food by the mouth, and feeding entirely by nutrient enemata; fomentations and poultices to abdomen; morphia hypodermically; while tincture of iodine, ipecacuanha wine, and tincture of opium in small and frequent doses were all tried successively by the mouth. In spite of treatment the vomiting continued almost incessantly for five days, being so continuous as to almost prevent sleep; the vomit contained bile, and occasionally small quantities of dark blood. The general nature of the case with the history of hematemesis, of course, strongly suggested gastric ulcer; but as the patient was extremely

emotional, and the vomiting was unusually frequent, and did not respond to treatment applicable to gastric ulcer, it was suggested that there was a marked nervous element to be dealt with. The patient was accordingly put on drachm doses of potassium bromide by the rectum four times a day, and in thirty-six hours the vomiting, which was previously increasing in severity, had entirely ceased. In four days more she was successfully taking solid food by the mouth, and in a fortnight was discharged cured.

She remained in good health a year, when the pain and vomiting suddenly returned, and she was re-admitted on January 28, 1898. The symptoms were exactly the same as in the previous year; the vomiting being almost incessant, the vomit consisting of clear, bile-stained liquid, frequently containing traces of blood. In this attack, however, the treatment by rectal feeding, bromide and morphia was absolutely inoperative. The vomiting increased, the pulse became rapid and feeble, and in twelve days the patient died, the end being accelerated by an attack of uncontrollable diarrhea with offensive stools.

The necropsy revealed absolutely no lesion except a catarrhal inflammation of the lower end of the ileum, which, no doubt, accounted for the terminal diarrhea. The stomach was perfectly healthy, except for a few submucous hemorrhages. There was no ulceration, erosion, or atrophy of the mucous membrane. The liver, kidneys and other organs were all healthy.

It is not stated that the nervous system was examined, and apparently no microscopic examination was made at all. PATRICK.

96. EIN EXPERIMENTELLER BEITRAG ZUR FRAGE DER PERIPHEREN DEGENERATIVEN NEURITIS BEI TUBERCULOSE (An Experimental Contribution to the Question of Degenerative Peripheral Neuritis in Tuberculous Persons). Carl Hammer (*Deutsche Zeitschrift für Nervenheilkunde*, 12, 1898, p. 215).

Neuritis has been so frequently found in persons suffering from tuberculosis that there must be some relation between the two diseases. Hammer found that he was not able to produce neuritis in guinea pigs, in every case, by the injection of cultures of the tubercle bacilli. When the material for inoculation was taken from a tuberculous human peritoneum the results were very different, and the nerves were found markedly degenerated. The material from the human peritoneum seems, therefore, to be more virulent. He found, also, that tuberculosis in guinea pigs, produced by any form of inoculation, invariably causes changes in the spinal motor cells of guinea pigs, detectable eight days after the inoculation, and in some cases sufficiently intense to cause death of the cells. Hammer believes that the neuritis found in man in tuberculosis and other infectious diseases is probably secondary to changes in the cells. SPILLER.

97. UEBER DAS WESEN UND DIE ENTSTEHUNG DER HEMIPLEGISCHEN CONTRACTUR (Concerning the Nature and Origin of Hemiplegic Contracture). Ludwig Mann (*Monatsschrift für Psychiatrie und Neurologie*, Vol. IV, Nos. 1 and 2, pp. 45 and 123).

The theories which are used to explain contracture by the loss of the inhibition transmitted through the pyramidal tract, or by the stimulation of the cells of the anterior horns from secondary degeneration of these tracts, are very unsatisfactory. In hemiplegia the paralysis and contracture affect distinct groups of muscles. The muscles

which are completely paralyzed are not contracted, and their passive mobility is exaggerated; the antagonizing muscles of these groups, however, have preserved their voluntary movement relatively well, are contracted. Mann suggests that possibly the excitomotor fibers of each muscular group are closely associated, or identical, with the inhibitory fibers of its antagonizing group. In the lower extremity in hemiplegic cases the contracture exists in those muscles which extend the limb, which "lengthen" it, and in these muscles power is relatively well preserved. In the upper limb the extensors are paralyzed and the flexors are movable voluntarily to a certain degree. The hemiplegic contracture is not the same as the contracture from peripheral cause. The hemiplegic contracture is due, not only to the innervation of the relatively well preserved antagonizing muscles, but also to the destruction of the inhibitory fibers to these same antagonizing muscles. Hering and Sherrington found that inhibition—recognized by flaccidity of a previously contracted muscle—could always be obtained by feeble irritation of that part of the cortex from which stronger irritation caused contraction of the antagonizing muscles; for example, when feeble irritation of the cortex caused relaxation of the biceps, stronger irritation of the same part caused contraction of the triceps with relaxation of the biceps. Hering and Mann say that the contraction of a muscle is usually associated with relaxation of its antagonist, contrary to the view of Duchenne, who held that in every contraction of a muscle a simultaneous contraction of its antagonist occurs, enabling the latter to act as a moderator in the movement. It is true that when a limb is held firmly in a certain position the antagonists are also contracted.

When one of two groups of muscles which act together is paralyzed peripherally, the other group is thrown into excessive action by voluntary attempts to innervate the paralyzed group; thus, when the extensor digitorum communis is paralyzed, attempts to extend the fingers have been found to cause flexion at the wrist joint; or when the interossei and flexors of the fingers are paralyzed, attempts to innervate these paralyzed muscles have caused extension at the wrist joint. In these cases the antagonizing muscles are not innervated in attempts to control the paralyzed muscles; for example, the fingers are not extended by efforts to flex the fingers when the flexors are paralyzed, which would be the case if Duchenne's views regarding the simultaneous innervation of opposing groups of muscles on voluntary movement were correct.

The simultaneous innervation of associated groups of muscles is probably of cortical origin.

When a limb is cut off entirely from its cortical centers, as seen in transverse lesions of the cord, the paralysis is flaccid. In rare cases of hemiplegia the arm is completely paralyzed; in these cases the flaccidity is complete. Secondary rigidity in hemiplegia develops simultaneously with the restoration of a certain amount of power in the paralyzed limbs. In rare cases of hemiplegia the muscles which have retained a certain amount of movement do not exhibit hypertonia; in such cases possibly the spinal cells or centripetal tracts are affected.

SPILLER.

98. A FORM OF NEURALGIA OCCURRING IN CYCLISTS. W. H. Brown (Brit. Med. Jour., February 26, 1898).

The author describes a condition of extreme pain and tenderness of the perineum, scrotum, testicles and sometimes of the penis and inner surface of the thighs, that occurs after prolonged, and especially

after very rapid, bicycle-riding. At times there is anesthesia of the parts instead of hyperesthesia. An entirely analogous trouble has been observed in women. The affection may be sufficiently severe to confine the patient to his bed for a month, but the term "neuralgia" would seem to be a misnomer for such cases, as there may be visible signs of bruising, with extravasations of blood. The treatment is at first absolute rest and later the adoption of a suitable saddle and moderation in the use of the wheel.

PATRICK.

99. NOUVEAUX FAITS RELATIF A L'ÉTUDE DES NEVRITES PERIPHERIQUES DANS LEUR RAPPORTS AVEC LE RHEUMATISME CHRONIQUE DEFORMANT (New Facts Relative to the Study of Peripheral Neuritis in its Relation to Chronic Rheumatism). Pitres et Carrière (Archives Cliniques de Bordeaux, No. 8, 1898, p. 408).

After considering the relations between chronic rheumatism and affections of the nervous system, and reviewing the results of examination of the cord and nerves in this disease, as reported by different observers, the authors proceed to give the clinical histories and the results of post-mortem examination in two very typical cases of chronic rheumatism, with much deformity.

CASE 1. Woman of forty years. Family history, negative. Personal history, negative, except that her work as a servant caused her to be a good deal exposed to cold and dampness.

The joint affection began in the left foot, and gradually proceeded until all the joints of the limbs except the hips, and even the jaws were affected. There was no sensory disturbance, except slight hypesthesia over the affected articulations. Such reflexes as were independent of the joint ankylosis were normal. There were no vasomotor disturbances. The skin and nails showed some trophic changes, and there was extensive muscular atrophy. In the apices of the lungs there were the physical signs of tuberculosis.

The patient dying, the autopsy revealed advanced tuberculosis of the lungs.

The affected joints showed thickening of the connective tissue, increase in size of the ends of the bones, eburnation and destruction of the cartilages in places, and development of vegetations on the synovial membranes. Pieces of nerves from different regions were hardened in 1 per cent. osmic acid solution and double stained with borax carmine. The cord was hardened in Müller's fluid. The anterior and posterior spinal nerve roots were normal. The large nerves of the arm showed an increase of fine, badly staining fibers, and, in places, segmentation of the myelins, and nuclear proliferation, more marked towards the periphery, the changes being found in their greatest intensity in the filaments supplying the fingers.

The sciatics on both sides were normal, but their branches supplying the knee-joints showed similar changes to those found in the nerves to the fingers. The lesions were those of parenchymatous and interstitial neuritis.

The cord was normal.

CASE 2. Woman of fifty-seven years. Family history, mother and grandmother rheumatic. Previous personal history negative.

The disease began in the small joints of the fingers.

The phalango-phalangeal and metacarpo-phalangeal articulations were all affected, the hands being much deformed, and being maintained in the position of flexion. There was great atrophy of the small muscles of the hand, especially of the interossei. The carpo-

carpal, and radio-carpal articulations were intact. The knee-joints could be flexed only with difficulty, the ends of the bones entering into them being much enlarged and deformed, and the neighboring muscles atrophied.

The other joints were normal. The skin about the affected articulations showed trophic changes, as did also the finger nails. The patient complained of severe pains, and of sensations of burning, and of cold, about the affected joints, but presented no subjective sensory symptoms, other than slight hypesthesia on the inner side of the knees. She developed a serous pleurisy, was tapped four times, but gradually ran down, and died six months after coming under observation. The autopsy showed slight fatty degeneration of the heart and of the liver, thickened pleura, and some fibroid changes in the lungs. The brain, cord and spinal nerve roots were normal.

The articulations of the knee-joints and of the fingers showed the characteristic changes.

The cord and nerves were prepared in the same way as in the former case, and were examined, with the following results: The cervical nerve roots, and nerves of the arm, were normal, the smaller branches to the fingers all showed degeneration, to a greater or less degree, as did also the filaments to the knee-joints and the sciatics in the lower part of their course. The lumbar nerve roots were normal, cord normal.

ALLEN.

PSYCHIATRY.

100. GENERAL PARESIS IN WOMEN. J. Hladik (Casopsis ceskych lékarn, 1898, p. 29). *Revue Neurologique*.

The results of the author's studies of general paresis in the asylums of Dobrau, in Bohemia, lead him to the following general statements:

(1) There has been no appreciable variation in the number of female paretics.

(2) The general average is one case in women to three cases in men.

(3) Dementia paralytica commences usually between the ages of 40 and 50.

(4) The most important etiological factors are heredity, alcohol and social misery. This last factor is especially predominant in the larger cities.

(5) With these people the course of the disease is slow; maniacal states, as well as paralytic attacks are rare. Ideas of grandeur are not marked. Agitation and dementia predominate.

(6) Remissions are by no means infrequent, and are of appreciable length.

JELLIFFE.

101. LES LÉSIONS TABÉTIQUES DANS LA PARALYSIE GÉNÉRALE (The Lesions of Tabes in General Paresis). E. Rabaud (*Revue de Psychiatrie*, 3, 1899, p. 37).

The author shows that according to the microscopical examination of the cord of cases in which tabes and general paresis are both present, there are grounds for separating the paralytic sclerosis from a tabes sclerosis. He believes that an essential point is the examination of the entire cord, at different levels, since at any one level the picture may resemble that of a posterior sclerosis very closely. The main general distinctions are: (1) The localization of the tabetiform

lesions varies at different levels of the cord. (2) Examination of any one section shows minute, yet striking differences in the fiber degenerations. (3) The posterior root-zone is usually healthy in general paresis. (4) The cells of the anterior horns and of the columns of Clarke are altered.

JELLIFFE.

102. CONTRIBUTION A L'ANATOMIE-PATHOLOGIQUE DE LA PSYCHOSE POLY-NEVRITIQUE ET DE CERTAINES FORMES DE CONFUSION MENTALE PRIMITIVE (Contribution to the Pathologic Anatomy of the Psychoses of Polyneuritis and of Certain Forms of Primitive Mental Confusion). Ballet & Faure (La Presse Medicale, 30 November, 1898).

The clinical histories and results of the microscopical examinations of the brains and cords of two cases of alcoholic polyneuritis are recorded. The first, a woman, thirty years of age, had been in the habit of using alcohol to excess for some years. After the death of a child, which caused her considerable grief, she refused to eat, appeared to have lost all energy, slept badly, and had hallucinations. Some three months later she suffered from darting pains in the legs, and there was edema of the lower extremities. When admitted to the hospital there was general weakness of the lower extremities of such an extreme degree that it was impossible for the patient to stand. The reflexes were exaggerated, and there was difficulty with the sphincters. She wept constantly, her knowledge of time and place was greatly disturbed, and she comprehended simple questions with difficulty. Memory was very imperfect. There were slight lesions at the apices of both lungs, but death appears to have been produced by an extensive bed-sore over the sacrum. At the autopsy the liver was found enlarged, the kidneys congested, the brain and cord apparently normal. Sections through the paracentral lobule showed extreme alteration of the large motor cells, not more than one in six being healthy. Many of the cells in the anterior cornua of the lumbar cord were likewise degenerated, showing the peripheral position of the nucleus, loss of the chromophilic bodies, etc. The anterior tibial nerve showed marked secondary degeneration. The second patient, a woman of thirty-two, had been drinking to excess for over a year, during which time she had also suffered from severe cough and hemoptysis. She remained quietly in bed, paid little attention to her surroundings, had difficulty in answering questions, and occasionally said a number of words without any particular connection. At the autopsy slight changes were found in the liver, the brain and cord appeared to be normal, but the cells in the paracentral lobule were degenerated. The cells of the anterior horns of the lumbar region were apparently normal. No changes were found in the white substance of either the brain or the cord by the methods of Pal or Marchi. A few degenerated nerve fibers were found. The interesting feature of this case is the absence of any secondary degeneration in the nerves, indicating either a primary affection of the cells of the cerebral cortex, or else their reaction to a degeneration at the periphery of the neuron, without consecutive change in the intervening portions. The authors are convinced that the alterations in the motor cells have a close relation to the psychical troubles. The changes in the cells were not similar to those produced by post-mortem putrefaction or by high fever. They call attention to the frequency with which polyneuritis, due to alcohol, is associated with mental confusion; Korsakoff having found that thirty of fifty cases were associated with mental changes.

SAILER.

THERAPY.

103. ON TENDON GRAFTING OR "FUNCTION TRANSFERENCE" IN THE TREATMENT OF INFANTILE PARALYSIS. Frederic Eve (*British Medical Journal*, No. 1972, p. 1139).

It may be laid down as a general rule that in selecting a muscle for grafting the one should be chosen whose action is most nearly allied to that of the paralyzed muscle or group, not only because such muscles are nearest, but also because restoration of voluntary function is more readily obtained. But the converse of this is sometimes, in the opinion of the author, advisable, and an antagonist of the paralyzed muscle may be selected.

The author reports several cases of localized paralysis from injury and from poliomyelitis, in which the results of operation were very good.

PATRICK.

104. TETANOS TRAUMATIQUE (Traumatic Tetanus). (Treated and cured by the intra-cerebral injection of anti-toxin. Method of Roux & Borrel, Chauffard & Quénu) (*Presse médicale*, June 18, 1898, p. 325).

The patient, a frail boy of 16 years, received a severe injury to the left hand, crushing one of the fingers. Four days later the hand was found to be swollen and edematous. There was no fever and little pain. Fourteen days after the injury there was some stiffness of the lower jaw, and the next day the patient was unable to open the mouth. There were pains in the neck. As the condition was growing rapidly worse, the patient received 20 cc of serum hypodermatically. Eighteen days after the injury, as the patient was beginning to have severe convulsions, the skull was trephined on both sides under strict antiseptic precautions, a needle thrust into the brain substance, and from 1½ to 2 cc. of anti-tetanic serum injected into the brain substance. At the same time the hand was thoroughly disinfected. One hour later the patient recovered from the chloroform narcosis and appeared perfectly rational, and there was already considerable improvement. From this time, although the tetanic symptoms continued, their force rapidly decreased, and eighteen days later the cure was complete. The results in this case correspond closely to the experimental results obtained by Roux and Borrel, who observed that the symptoms persisted for some time after injection.

SAILER.

105. NOTE ON THE ACTION OF BROMIDE AND IODIDE OF STRONTIUM ON EXOPHTHALMIC GOITRE IN CHILDREN. A. Lockhart Gillespie (*British Medical Journal*, No. 1971, p. 1042).

The author reports on the administration of ten grains of strontium iodide and five grains of strontium bromide t.i.d. to three deaf mutes aged eleven, ten and nine years respectively, who presented enlargement of the thyroid and tachycardia. Exophthalmos, von Graefe's and Stellwag's signs were absent. In each case considerable improvement was noted, the decrease in the circumference of the neck averaging 1-10 inch per day, for the first ten days of treatment.

PATRICK.

Book Reviews.

LES VOIES CENTRALES DE LA SENSIBILITÉ GÉNÉRALE, ÉDOUARD LONG.
G. Steinheil, Paris, 1899.

This thesis is published from the laboratory of Dr. Dejerine at the Salpêtrière, and represents largely the views held by this distinguished French neurologist. The subject which Dr. Long has chosen is not an easy one, and even after the thorough treatment it has received at his hands, we must acknowledge that much remains to be discovered. Dr. Long divides his book into two parts; the first contains a careful review of the literature, the opinions of the most distinguished investigators in regard to the sensory tracts, and the writer's own views; the second contains the reports of cases studied clinically and microscopically by the author. It would be a difficult and unnecessary task to review the statements abstracted from the literature, and it is more suitable to refer to some of Dr. Long's own conclusions.

Dr. Long does not think that the decussation of sensory fibers within the spinal cord has been fully established by physiological and pathological investigations; of course, he is not referring to the well recognized decussation of the posterior column fibers in the medulla oblongata. The symptom-complex of Brown-Séquard exists, but the anatomical explanation of a decussation of sensory fibers is hypothetical.

The theory of the individuality of tactile, painful, thermic and muscular sensations, and of the multiplicity of the organs of perception and transmission, is not in harmony with the results of physiological and anatomical study. The different impressions called sensory are created by peripheral excitation more or less intense.

Dr. Long believes that the median fillet terminates in the lower and external part of the thalamus, and does not pass directly to the cerebral cortex. If it did pass uninterruptedly to the cortex, it would undergo a retrograde degeneration almost as great from extensive cortical lesions as from thalamic, and this is not the case. Cortical lesions cause only a slight diminution in the size of the fillet, explainable by the degeneration of its accessory fibers. Cortical lesions, congenital or acquired very early, cause asymmetry of the fillet, and this asymmetry results from simple atrophy or arrest of development. Aberrant fibers unite with the fillet, and some descend into the medulla oblongata, but do not take part in the sensory decussation, and enter the pyramids; others terminate in the upper part of the pons. These accessory fibers are not the central tracts of sensory cranial nerves, nor is their only function the innervation of the nuclei of motor nerves. Their termination is unknown.

Dr. Long follows the teaching of Dr. Dejerine very closely. The thalamus, he tells us, is in connection with the entire cerebral cortex by means of the cortico-thalamic and the thalamo-cortical fibers, *i. e.*, by fibers passing from the cortex to the thalamus and *vice versa*. The views of Flechsig are not accepted.

No distinct sensory tract exists in the posterior segment of the

internal capsule, but on the contrary, the sensory fibers are mingled with the motor in the genu and the posterior limb of the internal capsule. Dejerine, some years ago, disputed the correctness of the views held in regard to Charcot's "carrefour sensitif." Long reports two cases in support of his statements; in these cases disturbance of sensation was not noticed. In the first the posterior segment of the inner capsule was destroyed and the thalamus was intact; in the second the lesion involved only a part of the posterior segment of the internal capsule, and only the anterior and internal nuclei of the thalamus, and not the external nucleus. Persistent hemianesthesia is more common when the thalamus is destroyed than in lesions of other parts of the cerebral hemisphere. The anesthesia from a cortical lesion is never complete, and diminishes in intensity after a time. Motility, general sensation and muscular sense have the same cortical localization.

The results of forty-one clinical observations and of thirteen clinical and pathological studies form a fitting termination to this valuable book. The name of Long may worthily be placed by the side of those of Vialat, Sottas, Mirallié and Thomas, who, under the inspiration of Dejerine, have made most valuable contributions to neurological literature.

SPILLER.

A MANUAL OF MODERN SURGERY, GENERAL AND OPERATIVE, by John Chalmers DaCosta, M.D., Clinical Professor of Surgery, Jefferson Medical College, Philadelphia; Surgeon to the Philadelphia Hospital, etc. With 386 illustrations. Philadelphia, W. B. Saunders, 925 Walnut street, 1898.

It is with pleasure that we note the appearance of a second edition of this most estimable manual. No other work on surgery we are acquainted with seems so well adapted to fill the requirements of that debatable ground which lies between the comprehensiveness of the full-fledged text-book or system and the conciseness of the quiz compend. Adequate descriptions are given of all the important surgical conditions with their treatment, separate sections being devoted to the operative technique, and a summary of the instruments needed. In this edition the advances of surgical knowledge have been fully recognized, among others, sections having been added on the Surgery of the Liver and Gall-Bladder, the Spleen, the Pancreas, the Female Breast, Wounds inflicted by Modern Projectiles, Electrical Injuries, and the Use of the Röntgen Rays. The following operations have been described: Resection of the Gasserian Ganglion, Methods of Gastrotomy, Schede's Thoracoplasty, Use of the Murphy Button, Various New Methods of Enterorrhaphy, Bodine's Method of Colostomy, Prevention of Hemorrhage in Hip-joint Amputation by Macewen's Method of Aortic Compression; Edmund Owen's Operation for Harelip, Senn's Method of Resection of the Shoulder-joint, etc.

The character of the illustrations and presswork is excellent, and the work is one to be heartily commended to all surgical students.

VOGEL.

LEITFADEN DER ELECTRODIAGNOSTIK UND ELECTROTHERAPIE. Für Praktiker und Sturierende von Dr. Toby Cohn, Berlin. Mit Einem Vorwort von Prof. Dr. E. Mendel. S. Karger, Berlin, 1899.

It is characteristic, we believe, of modern therapeutics to utilize more and more of physical methods for the treatment of disease. The influences of heat, light, massage, and electricity have been more carefully studied of late than ever before, and special therapeutic journals

devoted exclusively to treatment by diet, heat, light, massage, hydrotherapy, and electricity are more numerous now than formerly. The appearance of this guide to the use of electricity in diagnosis and treatment is therefore specially opportune. In 130 pages the subject matter is thoroughly presented. It is concise, yet broad enough for practical purposes. The successive chapters treat of the physics of electricity, the general laws and physiological phenomena, methods of investigation, changes in the reactions of the muscles and motor nerves, the electrical study of the organs of sense and electrical sensibility, the general therapeutic applications, special methods of treating, and electro-therapeutic apparatus. An appendix on Franklinization closes this excellent small manual. We recommend it highly. It is well illustrated and printed.

JELLIFFE.

BOOKS RECEIVED.

"Progressive Medicine," by H. A. Hare, M.D. Vol. 1, March, 1899. Lea Bros. & Co.

"Sajous' Annual and Analytical Cyclopedia of Practical Medicine." Vol. 3. F. A. Davis Co.

"International Medical Annual, 1899." E. B. Treat & Co.

"Practical Materia Medica for Nurses," by Emily A. M. Stoney. W. B. Saunders.

"Atlas of the External Diseases of the Eye," by O. Haub, M. D. W. B. Saunders.

"Transactions of American Electro-Therapeutic Association, 1898."

"Nervous and Mental Diseases," by Archibald Church, M.D., and Frederick Peterson, M.D. W. B. Saunders, 1899.

"Léçons sur les Maladies Nerveuses." E. Brissaud. Masson et Cie, Paris, 1899.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

ON CERTAIN FUNCTIONAL (CHIEFLY HYSTERICAL) CON-
DITIONS OF THE EYE:*

A REVIEW WITH SOME PERSONAL OBSERVATIONS.

By B. SACHS, M.D.,

PROFESSOR OF MENTAL AND NERVOUS DISEASES IN THE NEW YORK POLYCLINIC;
CONSULTING NEUROLOGIST TO THE MONTEFIORE HOME, ETC.

Last spring I had the privilege of presenting before the ophthalmological section of the Academy a patient who was suffering from a functional paralysis of several ocular muscles. The patient and the theme he suggested possessed more than a passing interest. Do functional affections of the eye occur frequently; are they hysterical or merely simulated; and how can they be differentiated from similar organic affections? These were a few of the questions demanding a solution. With some diffidence I accepted Dr. Gruening's invitation to bring the entire subject before an assemblage of oculists and general practitioners, for the subject is beset by considerable difficulties, many of which can be overcome only by one better versed than I can claim to be in ophthalmic science. But I concluded to attempt the task, upon the reflection that neurologists evidently see the patients with functional eye conditions far less

* Read before the New York Academy of Medicine (meeting in charge of Section on Ophthalmology), December 1st, 1898.

frequently than do the eye specialists, and the assistance of the latter will be sorely needed if the neurologist's studies are to be brought to a successful issue. I am anxious to establish the truth in this matter, not eager, however, to become the high-priest of hysteria.

The subject is not altogether new, though it has appealed to a relatively small number of investigators. But the few have made themselves heard in vigorous and often vehement discussions. In 1878, Charcot¹ described at some length the visual symptoms of hysteria, with special reference to muscular derangement. He was followed by Féré,² Parinaud,³ Ballet,⁴ Gilles de la Tourette,⁵ and others; most of these French writers discussing the ocular affections as mere incidents in the course of a general hysteria.

In Germany, the French writings on ocular hysteria were received with considerable skepticism, and to this day many German authors cannot think of hysteria otherwise than in connection with simulation. A number of them have, however, taken up the cudgels in defense of hysterical eye troubles. Manz,⁶ Struempell,⁷ Löwenfeld,⁸ Ziehen,⁹ Oppenheim,¹⁰ Donath,¹¹ and a score of others have reported cases of this description. At a recent meeting of German alienists and neurologists, some of these functional diseases were the subject of a lively controversy, Moebius¹² defending the rather extreme view that there are no hysterical ocular palsies, and that ptosis even is never purely hysterical. But the majority were not in sympathy with this opinion.

Oculists have remained much more skeptical than neurologists. But no one who reads the monograph of Wilbrand

¹ Charcot, *Progr. méd.*, Jan., 1878.

² Féré *Gaz. médicale* 1881.

³ Parinaud, *Annal. d'Oculistique*, 1887; also vol. 96, 1886.

⁴ Ballet, *Rev. de Méd.*, 1888.

⁵ Gilles de la Tourette, "Traité clinique et thérapeutique de l'Hystérie," 1891.

⁶ Manz, *Berl. kl. Wochenschr.*, Nos. 2 and 3, 1880.

⁷ Strümpell, *Deutsche Zeitschr. f. Nervenheilk.*, 1892.

⁸ Löwenfeld, "Neurasthenie und Hysterie," p. 402, Wiesbaden, 1894.

⁹ Ziehen, *Eulenburg's Realencyclopädie*, Vol. XI., p. 334, *et seq.*

¹⁰ Oppenheim, "Lehrbuch," p. 738, *et seq.*, 2d ed.

¹¹ Donath, *Verein der Aerzte zu Budapest*, Nov. 28. 1891.

¹² Möbius, *Arch. f. Psych.*, Vol. 31, p. 504.

and Saenger,¹³ the product of a fortunate co-operation between skilled specialists, can doubt the occasional occurrence of a variety of hysterical affections of the eye. Schmidt Rimpler, in his recent monograph, in Nothnagel's system, is compelled to admit that some ocular affections are hysterical, though he is one of those who incline to the opinion that it is our first duty in every instance to prove that the hysterical patient is not also a malingerer.

Wilbrand and Saenger deserve especial credit for their attempt to show that ocular conditions which were supposed to be peculiar to hysteria occur also in other functional diseases of the nervous system, and are the expression of early functional exhaustion of the optic apparatus. Nervous asthenopia, which owes its origin to Beard, has been put on a firm basis by these German writers. Among British authors who have studied the subject we are discussing, Bristow and Gowers may be mentioned as the most prominent. American writers have contributed relatively little, although Prince,¹⁴ Harlan,¹⁵ Booth,¹⁶ S. W. Mitchell,¹⁷ Mitchell, and de Schweinitz,¹⁸ George J. Preston¹⁹ and Lloyd²⁰ have considered various aspects of this subject.

I have thought it best to speak of "certain functional conditions" only, in order to be able to limit the discussion to a few questions of greatest interest. There is some satisfaction in being able to relegate muscular insufficiencies, the various "phorias," and the mysterious influences they are supposed to exercise over functional nervous diseases, to the tender mercies of knowing and discriminating oculists. We will have none of it—at least not for the present.

Hysterical palsies of the ocular muscles and hysterical amaurosis are the two subjects to which special attention shall be directed. It will contribute to a proper understanding of

¹³ Wilbrand u. Saenger, "Ueber Sehstörungen," Leipzig, 1892.

¹⁴ Prince, *Amer. Jour. of Med. Sc.*, 1897, Vol. 113, p. 157.

¹⁵ Harlan, *Trans. of the American Ophthalmol. Assoc.*, Boston, 1885, p. 649.

¹⁶ Booth, *Trans. of Am. Neurolog. Ass'n.*, 1895.

¹⁷ S. W. Mitchell, *Am. Jour. of Med. Sc.*, 1893.

¹⁸ J. K. Mitchell and de Schweinitz, *Journal of Nerv. and Ment. Dis.* 1894.

¹⁹ Preston. "Hysteria and allied conditions," Philadelphia, 1897, p. 76.

²⁰ Lloyd. *Dercum's Text Book on Nervous Diseases*, p. 111.

the matter in hand if I may be allowed a few words on our present conception of hysteria. Neurologists have advanced far beyond the stage when medical men spoke of hysteria simply because the disease or the condition did not appear to be anything else, thus using the word frequently as a mantle to cover up their ignorance. It was the fashion of old to diagnose hysteria by exclusion. Nowadays we recognize the disease by symptoms so positive that we can assert that the condition must be hysteria and can be nothing else. The attempt to define the disease has not been altogether successful. It has led to a division of writers into several distinct groups. On the one hand, it is classified among the general neuroses which are characterized by a distinct, though functional, derangement of the entire nervous system, a derangement due primarily to increased irritability and weakness of the more important nervous centers. Its chief ally in this group would be neurasthenia. In the opinion of others, chiefly of the French school, hysteria is essentially a psychic derangement, depending upon the disturbed function of the highest cortical centers. Moebius would include under this term all morbid physical conditions dependent upon concept. A third group of writers are content to call hysteria a psycho-neurosis. I am inclined to the belief that while there is a strong psychic basis for the development of hysteria, and while many of the phenomena can only be explained upon the supposition of the abolition of cortical influence, the important part played by pure exhaustion should not be overlooked. The truth of this is brought home to us by the common experience of many hysterical patients who present distinct neurasthenic symptoms.

Whichever definition we may adopt, the fact cannot be denied that in hysteria there are abnormal perceptions and concepts; peripheral stimuli are often not perceived, as they are in the normal cortex; and the abnormal conditions of the highest nerve centers exert their influence over somatic states. Sub-conscious and non-volitional actions are less subject to hysterical derangement than are distinctly volitional and purposive movements. Individual ocular muscles are, as is well known, subject to the will, but the exquisite adaptation of

ocular movements to their special ends is less under the control of the will than other physical actions are, as is evident, among other things, from the fact that in the dissolution immediately preceding death the ocular mechanism is almost the last to suffer, and the ominous rolling upward of the eyes is often synchronous with the last gasp. On the other hand, the relation of the eye movements to emotional conditions is well known. The great artists of all ages from Raphael down have appreciated this fact. It would be surprising, indeed, if the emotional disease *par excellence* were not at times distinguished by ocular derangement.

When hysteria affects peripheral parts of the body we may have sensory or motor disturbances. Anesthesia or hyperesthesia, palsy or spasm, are the symptoms, varying according to the predominance of a condition of irritability or of weakness in the motor or sensory nervous system. Hysterical affections are so manifold that they may be said to simulate almost any organic disease. However widely we may differ as to the theories regarding the disease, we must be entirely agreed as to the chief symptoms. These are:

1. An altered mental state—above all, an increased emotional condition.
2. Palsies, with or without contractures.
3. Subjective and objective disturbances of sensation (hyperesthesias and anesthetics). The latter may appear under the guise of an hemianesthesia, often involving the special senses, or of a regional, segmental, or "patchy" anesthesia.
4. Hysterogenic zones.
5. Typical convulsive attacks, occurring at irregular intervals.

All these symptoms are variable, and are particularly subject to suggestion by others, or to auto-suggestion.

With reference to the hysterical conditions of the eye, it is most important to look for the more important stigmata of hysteria, since the diagnosis may depend, to a very large degree, upon the presence or absence of such indubitable hysterical phenomena. It is scarcely necessary to add the caution that an hysterical subject may develop an organic affection of the eye, and even the presence of hysterical stigmata does not

make for the diagnosis of a special hysterical affection, unless it is certain that no organic disease is impending. I am anxious to convey the impression that true hysteria is after all an infrequent disease.

General hysterical affections may be diagnosticated not only by the presence of the special stigmata to which I have referred, but also by an association with certain symptoms, and possibly by the absence of some, the presence or absence of which we know to be incompatible with the assumption of an organic affection. I have frequently had occasion in my clinics to lecture upon a young Russian girl who had developed atrophic paralysis of the right lower extremity, with the loss of reflex and changes in electrical reactions. In the right upper extremity she developed, some years later, a paralysis without atrophy and without electrical changes, but with marked regional anesthesia. The paralysis of the leg was due to poliomyelitis; the affection of the upper extremity was distinctly hysterical.

While hysteria may simulate many other diseases, and may exhibit innumerable symptoms, we may assert that deep reflexes are never lost in hysteria, and that electrical reactions are never markedly altered. Is there any special symptom or association of symptoms in ocular hysteria by means of which we may positively diagnose hysteria and exclude organic disease of the eye, or *vice versa*? Great variability of the symptoms is an argument in favor of the hysterical character of an ocular affection. A ptosis that comes and goes in relatively short intervals, an amblyopia that disappears one day and returns the next, or a field of vision that is contracted to-day and much enlarged to-morrow, all such conditions are likely to be hysterical. If any of these conditions becomes permanent or chronic, it is, on that account, no less hysterical than is a paraplegia, which often lasts months, sometimes years. An unusual combination of ocular symptoms is also suspicious; thus, in a case of my own, photophobia, with a partial external ophthalmoplegia, offered good reason to doubt the organic nature of the affection. A patient described by Janet exhibited an internal hemianopsia, asthenopia, spasm of convergence, monocular diplopia, hemimacropsia and hemimi-

crospia—an association of symptoms that no one would ever dream of attributing to organic disease. It would be still more desirable if we could point to a single ocular symptom which would positively militate against the diagnosis of hysteria pure and simple, as the loss of the knee-jerk does against hysterical paraplegia. The pupillary reflexes would seem to serve such a purpose. It is an action not subject to the will, and should by rights be beyond the influence of hysterical derangement. In my own experience, I have never seen an abolition of the light reflex in hysterical conditions, and it may be allowed that it is not impaired in the majority of functional states. But several reliable observers have reported a disturbed pupillary reflex in purely hysterical states.

Dujardin²¹ described the case of a girl, 24 years of age, subject to attacks of amaurosis. During such attacks he noted a paralytic mydriasis. If the observation be correct, this mydriasis must be considered to be independent of the amaurosis; but is it not possible that hysterical amaurosis may in some instances be due to the fatigue of the retinal elements, a supposition which cannot be discarded off-hand? Parinaud recorded a paralytic mydriasis in a case of hysterical ophthalmoplegia. Mendel²² reported the transitory and absolute loss of the light reflex in a hysterical girl; while Donath²³ described a similar condition in a teacher 24 years of age. In her the right pupil was dilated and would not contract during accommodation. After a while, the right eye would react, and the left eye would be disturbed in a similar fashion. In this patient there was also a continuous right hemianesthesia.

Wilbrand, whose careful observations are beyond doubt, records a spastic miosis, with sluggish reaction to light, in a typical hysterical patient. Schwarz,²⁴ though skeptical regarding all hysterical conditions, believes that the occurrence of a spastic miosis, in association with a spasm of accommoda-

²¹ Dujardin, *Journal de Sciences Méd. de Lille*, Vol. V., 1889, quoted by Wilbrand and Saenger.

²² Mendel, *Deutsche Zeitschr. f. prakt. Med.*, 1874, No. 47 (quoted by W. & S.).

²³ Donath, *Wiener med. Presse*, 1892, No. 1.

²⁴ Schwarz, "Die Bedeutung der Augenstörungen für die Diagnose der Hirn u. Rückenmarkskrankh.," Berlin, 1898, p. 83.

tion and convergence, has been established by the case of Galezowski.²⁵ I have been greatly impressed by the strictures of Schwarz, who contends that a paralytic and non-toxic condition of the pupil can be safely diagnosticated only if such a pupil responds to the action of pilocarpine or eserine. Nonne and Beselin²⁶ record a case of left-sided mydriasis with paralysis of convergence, in which they were careful enough to exclude the possible use of atropine. Féré²⁷ insists that the pupils contract during the first stage of an hysterical seizure and become dilated as soon as the general convulsive movements set in. From this view there are dissenting opinions, and my own experience would not lead me to agree with it.*

Whether or not the pupillary reflexes are subject to hysterical derangement,† some of the observations just referred to will not permit us to diagnosticate organic disease and rule out hysteria because the pupillary reflexes are altered. On the other hand, hysterical patients may develop tabes, or multiple sclerosis, or even dementia paralytica, in which case disturbed pupillary reflexes would have a far graver significance. Nor are hysterical subjects immune to syphilis; and Oppenheim has described the condition of an hysterical woman who exhibited complete immobility of the pupils, evidently due to cerebral syphilis. There are pitfalls innumerable. All the more reason, therefore, why the subject should receive the closest possible attention at the hands of the oculists. I have often claimed that the pupil should be studied most carefully with reference to the special information it may give regarding the presence of constitutional syphilis; and although other physical symptoms may be absent, its possible bearing upon the diagnosis of hysteria should supply a further incentive to its most attentive consideration.

Let us now pass to the discussion of other functional derangements of the ocular muscles. It has been intimated be-

* Since the above was written, Spiller has recorded a carefully observed case of hysterical hemiplegia with rigidity of the pupil (*Philadelphia Med. Journal*, January 14, 1899).

† Cf. also A. Westphal *Berliner kl. Wochenschrift*, 1897, pp. 1024, 1052.

²⁵ Galezowski, *Progr. méd.*, 1878, p. 39.

²⁶ Nonne and Beselin, quoted by Schwarz; *loc. cit.*, p. 84.

²⁷ Féré, *Gaz. méd.*, 1881, No. 50, p. 703.

fore that hysteria may produce either a spastic or paralytic condition. Among the former, the spasm of convergence is the most frequent. Internal squint is common enough, and needs to be differentiated from paralysis of the external recti by careful testing of the double images. Miosis and a spasm of accommodation may be associated with the former, but does not occur with paralysis of the rectus externus. Monocular diplopia and polyopia, micropsia and macropsia are recorded as occasional subjective accompaniments of this condition. The relation which these symptoms bear to the anatomical structure of the lens, you will not expect me to dilate upon. A paralysis of convergence has not been observed in hysterical patients, but in neurasthenics it has been recorded by Parinaud, who also notes the occurrence at the same time of a preserved reflex to light, while the reaction during accommodation was lost. I have the records of two exactly similar cases among my neurasthenic patients, but I was not able to interpret the condition until I have been enlightened by the observation of Parinaud. Conjugate deviation of both eyes is commonly observed in hysterical seizures. In one of my patients, a young girl of 18, such deviation to the left persisted for a full hour after the attack, and disappeared after a sound sleep. The seizure which I witnessed was not of an epileptic character. It is rare, however, for such deviation to persist for any considerable period of time.

Hysterical ptosis may be the result either of spasm or of palsy, spasm of the orbicularis palpebrarum, or palsy of the levator. Parinaud^{27a} is responsible for the term "ptosis pseudo-paralitique;" and Charcot has shown that in cases of apparent ptosis due to spasm, the eyebrow is elevated and not depressed as in general ptosis. The skin of the upper lid is thrown into folds and is not smooth as in the paralytic form. Fibrillary twitchings in the upper lid are common, although these occur much more frequently in neurasthenia than in hysteria. In my own experience, which is necessarily limited, hysterical ptosis is due as often to spasm as to palsy. I have been impressed by the factor of exhaustion in several of such cases, and cannot forego

^{27a} Parinaud, *Gaz. hebdomadaire de médecine*, 1877, Nos. 46, 47.

the opportunity of mentioning the resemblance of these hysterical affections to a ptosis which has recently been described as one symptom of myasthenia gravis, in which many of the muscles supplied by the cranial nerves show a weakness due to exhaustion. The majority of neurologists will surely not join Moebius in the belief that there is no such thing as hysterical ptosis. Oppenheim has recently stated that in hysterical paralytic ptosis the upper lid becomes retracted if the head is tilted backward, a symptom which will well bear further scrutiny.

In view of the frequent occurrence of ptosis in the early stages of tabes, in multiple sclerosis and in brain syphilis, the diagnosis between hysteria and these conditions should be established with the greatest care. I repeat that variability in the symptoms, the existence of other stigmata of hysteria, will be valuable aids in proving the diagnosis. Recurrence and relapses are, however, not unknown in syphilitic affections of the brain, more especially in those forms which involve the cranial nerves at the base.

The existence of an hysterical paralysis of the external ocular muscles has been warmly defended by French writers and by several German authors, notably Wilbrand and Saenger. But many others, including Mauthner,²⁸ Schwarz (*loc. cit.*) and Schmidt-Rimpler,²⁹ concede that such palsies may occur, but contend the case has not been sufficiently proved. A short synopsis of a well authenticated case by Wilbrand and Saenger will give you the facts. It remains for you to endorse or dissent from the diagnosis:

A young lady, 25 years of age, had been subject to spells of crying and weeping at the age of puberty, and had suffered from a number of nervous disturbances. At the age of 20, she began to experience trouble with her eyes, which was not relieved by glasses. At the age of 23, Wilbrand made out an isolated paralysis of the inferior oblique of the left eye, with all its characteristic symptoms. The false image was above the true, the upper end of the former inclining laterally. The distance between the double images increased with adduction.

²⁸ Mauthner, *Deutsche Arch. f. kl. Med.*, XXI.

²⁹ Schmidt-Rimpler, "Die Erkrankungen des Auges, in Nothnagel's, *Specielle Pathologie*," etc., Vienna, 1898, p. 256, *et seq.*"

Prisms brought relief for a time. After relapse of a year, the same patient exhibited an isolated palsy of the superior oblique of the right eye. At this examination Wilbrand also noted a moderate contraction of the visual fields. Left vision was 20-50, right vision, 20-30. The pupils and the fundi were normal. In addition, there was marked anesthesia of the right half of the body, more especially in the right arm, in the hand and in the head. Treatment by powerful suggestion brought about the cure of the palsy in a few weeks, while the sensory disturbances and the concentric limitation of the visual fields persisted.

Schwarz, in criticising this case, is dissatisfied because it took several weeks to cure the patient. Suggestions need not work instantaneously; moreover, the sensory symptoms persisted in this case because they were in all probability not made the subject of suggestion. The cases of Parinaud, Borel and Nonne will also pass muster as typical hysterical ocular palsies.

I would not worry you with detailed histories, but let me review briefly the condition of a patient whom I presented hurriedly some months ago.³⁰ After a more thorough acquaintance with the literature of the subject, I find that our patient presents a number of specially valuable symptoms. In no other case do I find so clear a history of the sudden and emotional onset of the disease, a mode of origin which seems to me to argue largely in favor of the hysterical nature of the disease. The patient was kindly referred to me by Dr. Marple, to whom I am also indebted for the careful ophthalmoscopic examination and for the charts of the visual fields:

The patient—F. S.—is 51 years of age. For many years he has been a heavy drinker of beer; also gives an uncertain history of previous specific infection. He has been married during the past 27 years. His first wife, who is said to have had a left ocular palsy, died from a paralytic stroke about seven years ago. Six years ago he married his second wife. The patient led a regular life; attended to his business as a liquor dealer. At one time he weighed 300 pounds; now he weighs 230 pounds. He was in good health until January 12th, 1898. On that day he attended the funeral of a friend. While standing on the sidewalk, waiting for the coffin to be carried out,

³⁰ This Journal, 1898, p. 452.

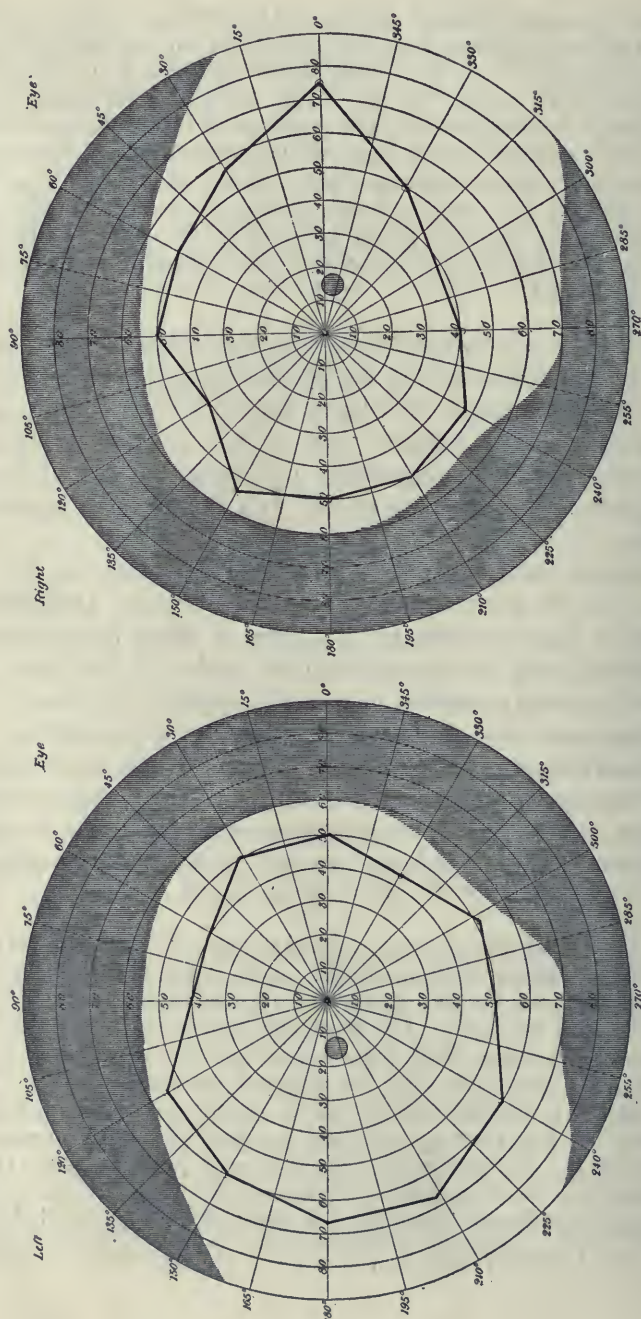


Fig. I.—Case of Marple and Sachs; fundus normal, as determined in Nov., 1898.

he noticed a flash of light before his eyes, began to see double, felt slightly dizzy, and could not look into the light. At no time did he lose consciousness. He was badly frightened; knew that he had the same trouble from which his first wife suffered, and of which she died. Some friends assisted him to his home. On his arrival, he found that both eyelids drooped; the photophobia was intense, and this symptom was the most

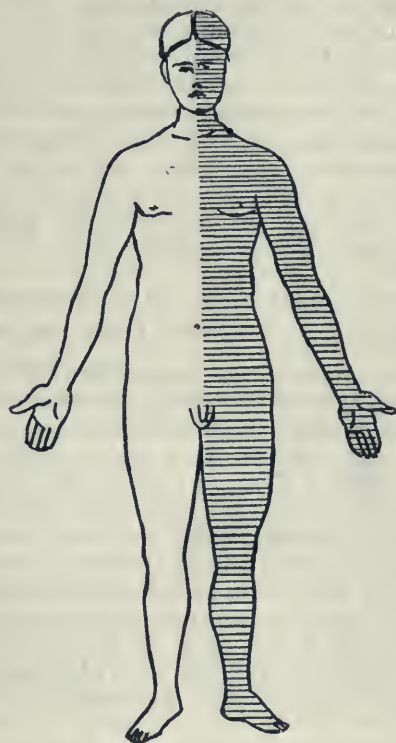


Fig. II.—Case of Marple and Sachs; functional ophthalmoplegia; left hemianesthesia and hemianalgesia.

distressing for many weeks. At the time of my first examination (March 5, 1898), the following record was made:

Double ptosis. When asked to look up, makes a great but unavailing effort; slowly raises the entire head, but does not use the frontales muscles, as patients with ptosis ordinarily do. After repeated commands, manages to raise the eyelids a little, but does not do so again, though requested to do so frequently during the course of a long examination. On lifting the eye-

lids with the fingers, it is seen that the axes converge slightly. Conjugate movement of the eyes to the left is imperfect; to the right the movement is performed easily. Upward and downward movements of both eyes are slightly limited and done in a jerky manner. Testing the eyes singly, it is noticed that the l. rectus externus moves the eye about half its normal distance. All other movements are performed more satisfactorily; but the movements are jerky, and at times succeed very much better than at others; muscles appear to be easily fatigued. The pupils are slightly irregular in contour; they react, though feebly, to ordinary light, but contract promptly during accommodation. The consensual reflex good. Dr. Marple reports that there are "no fundus changes. There is some apparent contraction of the visual fields. He has no central color scotoma, and his visual fields for color show no marked peculiarity. He has some choroidal changes, such as are usual in myopes. He also has some astigmatism.

Left vision is 20/40; with -1 D. cyl. ax. 90=20/30.

Right vision is 20/40; with -1 D. cyl. ax. 90=20/30."

A complete examination of the patient did not reveal any palsy in any other part of the body; the reflexes were normal, and there was no ataxia. Gait and station were normal. The head was held in a rigid position, with a slight curve to the right, from an evident desire to avoid the light. The only other symptom was a complete left hemianesthesia, with the exception of the left cornea, which was sensitive to touch. In every other part of the left half of the body, including the left nostril and the left half of the tongue, severe pricks with a pin were not felt. Touch, temperature and muscular sensations were normal in both halves. In the further progress of the case (to May 1, 1898), few changes were observed until after an attempt had been made to effect the trouble by hypnotic suggestion.

A few weeks ago I sent for the patient again, and found that his condition had changed considerably; that the ptosis had almost entirely disappeared, and that he could move his eyes very much more freely in all directions. The pupils reacted promptly to light, and he had not been troubled with double vision since the time of the last examination; the photophobia had disappeared, and the hemianalgesia had also improved to such an extent that he was only a little less sensitive to the pin test on the left side than on the right. While attending a picnic during the summer months, he stumbled and was severely shocked. To this accident he attributes the improvement, and not to the previous treatment. I will not dispute

this theory, and am satisfied to record that another emotional incident was responsible for the diminution of his symptoms.

In view of the alcoholic and possible specific history of the patient, it was natural to think first of the possibility of an organic lesion; but the sudden origin of the palsies, amid emotional excitement, the unusual variability of the symptoms during a single examination, the palsy of movements rather than of single muscles, the preservation of pupillary reflexes, the normal functioning of several muscles supplied by the third nerve, in spite of the marked ptosis, militated against the diagnosis of an organic ophthalmoplegia. Moreover, no organic lesion could account for the intense photophobia in the absence of all disturbances of the external eye. The contraction of the visual fields was slight, but sufficient to corroborate the diagnosis. The presence of hemianalgesia, and the improvement, either by suggestion or by an emotional accident, argue also in support of the diagnosis of a functional partial ophthalmoplegia.

Up to this time we have discussed only motor affections of the eyes, supposed to be due to hysteria. It now remains to consider sensory affections. Among these, the most important are the concentric limitation of the visual fields, the disturbance in perception of colors, and, above all, the forms of amblyopia and amaurosis. A few facts, safely beyond dispute, shall be briefly mentioned.

The concentric limitation of the visual fields is one of the most constant symptoms of hysteria—in fact, so common a symptom that the diagnosis of the major disease is often based upon it. Dana,³¹ among others, considers the contracted visual field to be the most frequent stigma of hysteria. It is not, however, pathognomonic of this disease, for it occurs in other conditions, notably in various forms of traumatic neuroses, which are not always mere varieties of traumatic hysteria.

According to Parinaud, central vision is never diminished, however narrow the field may be; while Wilbrand believes that the acuity of vision is generally diminished in these hysterical forms of retinal anesthesia. There is complete agreement as to the symmetrical character of this special visual disturbance.

³¹ "Textbook of Nervous Diseases," 4th Ed., p. 480.

Charcot's claim that limitation is greater on the side of the tactile anesthesia has been copied by many writers, but has not been corroborated by a majority of those who have taken the trouble to investigate the matter for themselves.

The color fields in hysteria are also limited and altered in a very peculiar fashion, and not necessarily in the proportion to the amount of concentric limitation. Green and violet are, as a rule, the first to be lost, but red is often recognized when blue is not. I have under observation at the present moment a young hysterical woman who has ample visual fields, but has lost all appreciation of colors, except for red and white. Wilbrand insists that the preference for red is not characteristic of hysteria, and that it occurs in neurasthenia and in other general neuroses. Up to the present time I have not been able to confirm this statement.

In addition to the concentric narrowing of the visual fields and to the changes in perception of color, there may be every other possible form of visual field defect in hysteria. Freud, as late as 1893, expressed the opinion that hemiopic defects have not yet been described in hysteria, and never will be. Lasting hemiopia is surely an extreme rarity in hysteria. Janet's case, to be referred to later on, is much to the point, and it is well to note that Dejerine and Vialet³² have described a special form of functional hemianopsia in neurasthenia and traumatic neurosis. Nasal hemianopsia and other forms of irregular scotoma have been recorded. The very oddity of the visual defect is of itself suspicious of hysteria, but chiasm lesions should in such cases not be lost sight of. The association of an irregular or unusual visual defect, with other ocular anomalies known to be due to hysteria, may be considered to be confirmatory of the diagnosis. Janet's case, which was also examined by Parinaud, is most instructive. The patient was a woman, 42 years of age, with marked sensory changes in the right half of the body, who complained of not being able to see objects except such as were to the left of her. The perimetric examination showed a concentric limitation of both fields, more pronounced in the right, with obscuration of the nasal half of the field. The same patient exhibited micropsia (probably due to a

³² *Comptes rendus de la Soc. de Biologie*, July 28, 1894.

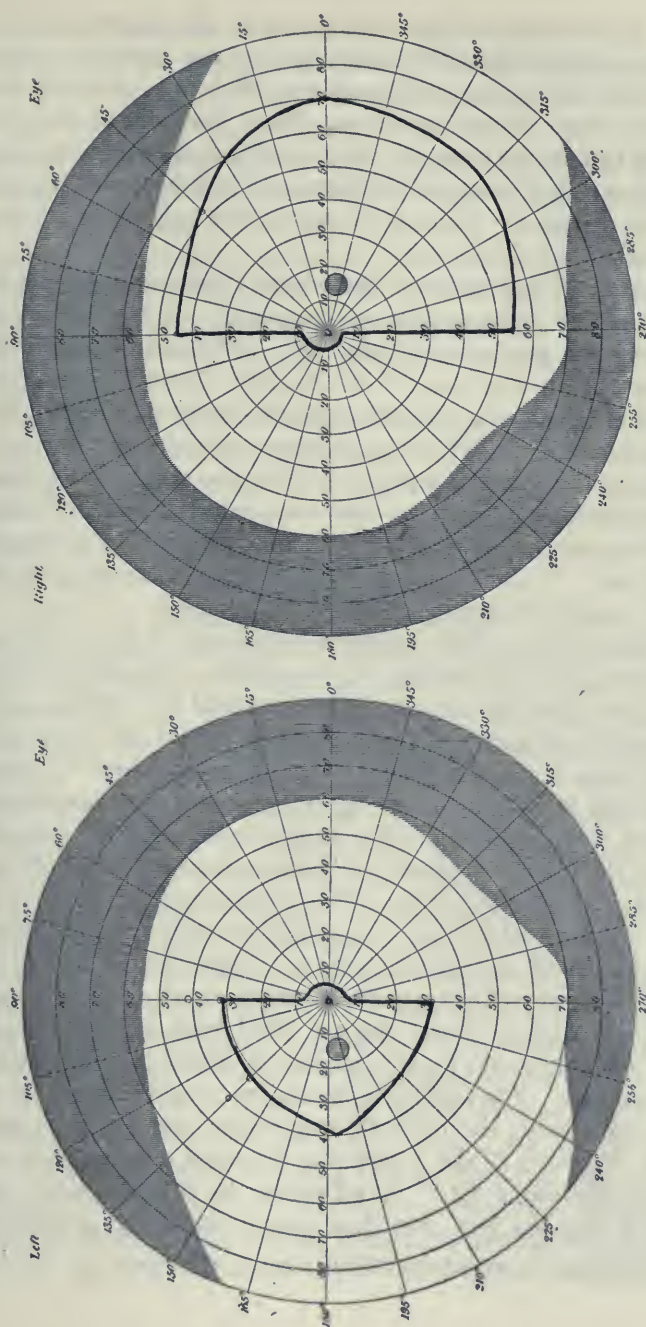


Fig. III.—Janet's case "Justine"; limits of the hemiopic visual field.

spasm of accommodation, to a spasm of the ciliary muscle), also deafness with complete anaesthesia of the same ear (externally); and the author, philosopher as he is, has much to say about the loss of binocular vision. From his point of view, the inability to fuse the image of one eye with that of the other being a return to a lower animal state. Of the hysterical nature of all the symptoms in Janet's^{32a} case there can be no reasonable doubt.

Concentric limitation of the visual fields may ultimately give rise to an amblyopia or an amaurosis. I am convinced, however, that only a few of the cases of hysterical amaurosis are developed in this way. It is especially characteristic of the latter that they come on with extreme suddenness, and often vanish as quickly. They are, however, not always transitory, and it is to the forms of more or less chronic amblyopia of hysterical origin that I wish to direct special attention; for these are the forms that have given rise, and justly so, to vehement discussion. If the diagnosis of hysterical amaurosis is to be upheld, several conditions should be considered:

First, the somewhat sudden development of the condition often amid, or subsequent to, emotional excitement. Secondly, the ophthalmoscopic findings should be negative, and must remain negative for a prolonged period of time; and, thirdly, even if these conditions be fulfilled, it is well to make certain that there is nothing else in the course of development, and that there are some other indisputable signs of hysteria present, though the presence of the latter need not preclude the possibility of organic trouble. The oculists have naturally suggested various tests for the purpose of detecting hysterical blindness—the use of the screen, “boîte de Flees,” etc. If hysterical patients recognize objects (under a peculiar form of stereoscope, for instance) which those who are blind from organic disease do not see, the former are not necessarily guilty of deception or of simulation. They do not perceive the retinal images; or, as Schweigger, with the oculist's reservation and mental smile, puts it, “they see if not aware that the blind eye has been engaged in the visual act.” At all events, let it be distinctly understood that the idea that they cannot see is the governing

^{32a} “Nevroses et Idées fixes,” Paris, 1898, p. 270, et seq.

and predominant factor. The blindness is for the time being as real to them as is the amaurosis of a tabic patient.

That complete hysterical amaurosis is rare is surely true. Several of my hysterical patients have made statements to me of having passed through a period of complete blindness after certain injuries, after the menstrual period, etc.; but I have actually seen it but once in a young girl of 18 subject to ophthalmic migraine, in whom a severe attack of sick headache was followed by an hour's absolute blindness. The hysterical character of this trouble was rendered probable by the fact that at the same time the girl exhibited marked segmental anesthesia.

Mendel (*loc. cit.*) recorded the case of a girl, 26 years of age, who was delirious and maniacal at times. There were no changes in the fundus. The pupils were variable in size and in their response to light. During eight months the girl was blind; so says the neurologist Mendel—a competent, impartial observer.

Mauthner, with the skepticism peculiar to oculists in this matter, accuses the girl of simulation, because she winked occasionally when the finger was near the eye; and Schmidt-Rimpler (*loc. cit.*, p. 259) thinks she may have been amaurotic for a time, recovered, and may then have simulated her former illness. Schmidt-Rimpler is less severe in his criticism of a case of Jacobson, which seems much more doubtful to me. This patient was an unmarried woman of 30, who became totally blind and remained so for several weeks. There was not even perception of light, and no reaction of the markedly dilated pupil; no retinal changes. That markedly dilated pupil would lead me to suspect either some deep-seated organic trouble or some drug influence. Oppenheim, who is skeptical enough as regards most medical questions, describes the disease of a patient in whom blindness lasted for months, and once over three years, and who had passed through thirteen attacks of blindness in ten years (*loc. cit.*, p. 738).

I would prefer not to accept as a fact the occurrence of complete hysterical amaurosis of months' duration until more convincing cases shall have been brought forward. We are apt to doubt such occurrences until satisfied by personal experience.

Such was my own attitude in former years with regard to hysterical ophthalmoplegia. However skeptical we may be with regard to complete hysterical amaurosis, the unilateral form is acknowledged by all. As a rule, these patients are as much surprised by the onset of this trouble as though they had been suddenly afflicted with organic blindness. Parinaud has shown that the amblyopic eye may not exhibit its defect in binocular vision. Herein was supposed to be evident proof that the unilateral amblyopia was sheer simulation, and nothing else. But this argument has been most clearly disproven by Morton Prince (*loc. cit.*), who found unilateral amblyopia in a patient who had just made an application for a position on the Boston police force, who had excellent vision with both eyes open, but when each eye was tested separately marked amblyopia was revealed. Putnam and Wadsworth vouch for the correctness of this observation.

We must bow to such evidence as this, unless the oculists will give some other explanation of such an occurrence. On the other hand, I am thoroughly in sympathy with those ophthalmologists who would not make the diagnosis of hysterical amaurosis, if there be the least reason to suspect either some form of toxic amblyopia, a retrobulbar neuritis, or an incipient tabes, or multiple sclerosis. I will take it for granted that a careful examination will be made to exclude the presence of these organic diseases, and, for my own part, would never give an opinion upon any case of visual defect without at least examining the deep reflexes and the reactions of the pupil. In tabes, optic atrophy is, as a rule, coincident with the development of sight defect, though the reverse is even more generally observed that there is only a very slight, or possibly no, visual defect for some time after the ophthalmoscope reveals incipient changes. It is well, however, to remember in this connection that a number of authors have recorded disturbances of vision in multiple sclerosis at a time when the ophthalmoscope revealed little or nothing. But at this early period of a multiple sclerosis other and sufficient signs would be found present. Above all things else, brain tumors should be ruled out before the diagnosis of hysteria is made.

There will be little confusion in attempting to make the dif-

ferential diagnosis between hysterical affections and the other diseases just mentioned, with the one exception of retrobulbar neuritis. It has been a surprise to me to find how little the writers on ophthalmology have to say on this subject; and yet, it is the one disease which may simulate hysteria most closely, for it gives rise to central and paracentral scotomata, to painfulness of the eye to touch, to photophobia, and to vague pains on movement of the eye muscles—all these symptoms being very much like those met with in pure hysteria. One distinction, according to Wilbrand, is to be found in the fact that the central and paracentral scotomata do not occur in functional diseases of the nervous system, but in the latter irregular and circular defects are much more common.

Leber,³³ a number of years ago, referred to the occurrence of peripheral optic nerve affections in hysterical patients. These patients exhibited marked unilateral amblyopia, dependent upon central scotomata, eventually leading to a white discoloration of the papilla. Higier,³⁴ of Warsaw, has detailed the condition of a patient who developed double optic neuritis after marked emotional excitement. The patient, a man 38 years of age, was totally blind, but recovered vision within a year under the influence of atropine and strychnine. Hysterical patients may develop retrobulbar neuritis, but it would be contrary to all our conceptions of hysteria if we were to consider this retrobulbar neuritis an integral part of the hysterical neurosis. Of all the questions which I have encountered in the course of my studies on this subject, none has puzzled me as much as the differential diagnosis of hysteria from retrobulbar neuritis, and the possible relation of the two forms to each other. It seems to me that oculists will do well to pay further attention to this special division of the subject.

Two patients, whom I have had occasion to observe with Dr. Marple, will serve to illustrate the difficulties to be encountered in the attempt to judge of ocular hysteria. These two patients presented marked visual disturbance, without perceptible changes in the fundus; yet it is doubtful whether either one of these cases can be claimed to be an example of hysterical amblyopia or amaurosis.

³³ Arch. f. Psychiatrie. Bd. 24, Heft., 2.

³⁴ Higier. Neurolog Centralblatt, 1898, p. 389.

The first patient was a man who had become suddenly amblyopic after having taken large doses of quinine, but the fact of his having taken this drug was ascertained only after much questioning, and some time after the first examination.

The second patient was one referred to my clinic, a few weeks ago, by Dr. Marple. A laundress, single, aged 52 years, woke up one morning to find herself totally blind in the left eye. There was no other subjective or objective symptoms. The patient is a thoroughly sensible, hard-working woman, presenting no characteristics of hysteria, and exhibiting not a single stigma; nor was the onset of the blindness preceded by any great emotional excitement. On this account, I was loath to make the diagnosis of hysterical amblyopia, and suggested the possibility of some retrobulbar trouble. All the pupillary reflexes, including the consensual reflex, are present. The total blindness seems, after a lapse of four weeks, to be receding, for the patient now has perception of large objects in a small portion of the nasal field. Dr. Marple is, I believe, inclined at present to the diagnosis of retrobulbar neuritis, as he has found a pallor of the optic disc. There is no clew to the etiological factor in this case.*

It was my intention originally to treat of a number of other functional conditions, particularly of nervous asthenopia, and to speak of the occurrence in neurasthenia of the same conditions which are observed in hysteria. But I fear that I have already exceeded the limits of this address and will, therefore, content myself with having shown that hysteria does occasionally involve the eyes, that it may produce motor and sensory ocular phenomena, and that these should be judged by the same standard which would apply to hysterical symptoms in other parts of the body. Hysterical ophthalmoplegia has been observed in a number of cases in which no other diagnosis could justly have been made, and the presence of general hysterical symptoms in these cases lends color to the diagnosis. The hysterical character of these ophthalmoplegias is proved, moreover, by the presence or absence of symptoms which

* In still another case in spite of the most careful and repeated examinations, by a most competent observer, a functional trouble was diagnosed until several months later the indubitable signs of brain tumor appeared.

would not be compatible with the assumption of an organic nuclear or root ophthalmoplegia. As for hysterical amaurosis, the unilateral form is confirmed beyond doubt, while there is some reason to doubt the occurrence of complete and chronic hysterical amaurosis. The relation of retrobulbar neuritis to hysteria needs further study. But I trust I have said enough to impress you with the fact that while I am not at all in favor of establishing the diagnosis of hysteria except on the safest possible grounds, it will not do to turn our faces against the possible occurrence of hysterical ocular affections for no other reason than that we have inherited since our earliest medical days a certain dislike and disdain for all phenomena of an hysterical nature.

Lastly, let me remind you that while hysterical patients may simulate, it would be a grievous error to make hysteria synonymous with simulation.

106. TUBERCULE PRIMITIF DE LA MOELLE: MÉNINGITE TUBERCULEUSE SECONDAIRE, TUBERCULOSE CONCOMITANTE DES GANGLIONS BRONCHIQUES, DE LA PLEVRE, DU POU MON, DU FOIE, DE RATE ET DU REIN DROIT (Primary Tubercle of the Spinal Cord: Secondary Tubercular Meningitis, etc.). Aniel et Rabot. (Lyon Médical. 88-1898, p. 605).

The patient was an infant nine months old when admitted to the hospital. A month before, the mother had noticed that the child no longer moved the left leg, and the physician consulted made a diagnosis of infantile spinal paralysis. On the morning of the day of entrance into the hospital the child had a convulsion lasting about ten minutes. Examination on entrance revealed very little except some general restlessness and agitation and the paralysis of the left lower extremity, but a diarrhea that had begun four days previously continued. In the paralyzed member there was some atrophy, loss of the knee-jerk, preservation of the plantar reflex, some contracture of the calf muscles and apparently normal sensation. The knee-jerk on the right side was exaggerated, sphincters normal. After admission the general condition became rapidly worse, one symptom of meningitis after another appeared, until at the end of a week the picture of this disease was complete and the child died nine days after admission.

The autopsy revealed a luxuriant cerebro-spinal tubercular meningitis, numerous enlarged and caseous bronchial glands, tubercles in pleura, lungs, spleen, liver and one kidney, and in the lumbar enlargement of the cord a tubercular mass, diffuent in the center. This agglutination of tubercles, or solitary tubercle, occupied the interior of the left half of the cord, the exterior appearing normal and the right side not invaded, which readily accounted for the monoplegia. The author thinks that the condition of this medullary tubercle indicated it as the point of primary infection and dissemination.

PATRICK.

OCULO-MOTOR PARALYSIS FROM TYPHOID FEVER, WITH A CASE.¹

BY G. E. DE SCHWEINITZ, M.D.

PHILADELPHIA.

That affections of the intraocular and extraocular muscles may occasionally complicate typhoid fever is a matter of record in medical literature. Thus, during the period of convalescence, dilatation of the pupil and paresis of accommodation are not uncommon, while mydriasis without cycloplegia and with normal vision, as Segal has pointed out, may be the result of irritation of the sympathetic.

Paralysis of the extraocular muscles is a much rarer phenomenon, and probably seldom occurs during the height of the fever in the absence of intracranial complications. Double ptosis and abducens palsy have been reported by Nothnagel in the third week of the disease.

During convalescence, according to ¹ nies, these extraocular palsies are more frequent, and he refers to certain varieties which quickly appear and quickly relapse, and which he believes to be nuclear and attributes to a chronic nephritis which he thinks is a frequent sequel of typhoid fever.

Finally, certain muscular paralyses have occurred at long intervals after typhoid fever and have been attributed by their reporters, for example, Runeberg, who has noted trochlear paralysis one and one-half years after the fever, to the typhoid poison. The etiological relationship, under these circumstances, it would seem, however, is rather far-fetched. Such cases as Samuel West has observed during mild attacks of typhoid fever, especially in children, which are characterized by strabismus and retraction of the head, and which disappear in a few days, may be the results of a mild basilar meningitis, or may be due to the effect of the poison acting with great intensity on the nervous system.

I have gathered these cases together in a chapter on the ocular complications of typhoid, and they with their references

¹Read before the Section of Ophthalmology of the College of Physicians of Philadelphia, April 18, 1899.

may be found on pages 312 and 313 of Keen's "Surgical Complications and Sequels of Typhoid Fever." To this list I desire to add the following case:

Mr. R., aged 22, born in America, was referred to me by Dr. C. S. Reynolds, and presented himself for examination on the 8th of March, 1899.

History—There is nothing in the family history of special interest, the father and mother both being healthy persons. One brother had a severe attack of typhoid fever associated with cerebro-spinal symptoms, leaving him partially hemiplegic for a number of months. On August 19, 1898, the patient was attacked with typhoid fever, which pursued an ordinary but somewhat severe course until September 12, when convalescence seemed to be established. On September 20 there was a relapse and the patient was suddenly seized with vomiting, headache and abdominal symptoms, the last of which, the attending physician tells me, suggested in their severity intestinal obstruction. At the end of the third day of this attack there developed complete right oculo-motor paralysis with ptosis. The patient slowly convalesced in spite of an attack of obstruction of the bowels on October 14, from which he entirely recovered. When I examined him in March of the present year, although still weak and slightly staggering in his gait, there were no marked symptoms of the severe illness from which he had suffered except the oculo-motor palsy.

O.D.—V. with $+1.25^{\circ}$ axis 90 was 6-6, accommodation was ample—0.50 pp. 14 cm. The eye was divergent, the angle of strabismus being 22 degrees; the outward movement was perfectly preserved; the inward rotation was most limited; there was also some limitation of upward and downward movements. These rotations measured on the perimeter were as follows: Outward 45 degrees, upward 20 degrees, inward to the median line, possibly 5 degrees nasalward, downward 40 degrees. Thus, there was loss of 10 degrees of upward rotation, practically the entire inward rotation and about 20 degrees of downward rotation. The pupil was round and contracted promptly to light stimulus and efforts of convergence. The optic disc was a vertical oval, of good color. The veins, however, were exceedingly full, especially the upper temporal vein, and there was slight venous perivascularitis. No splotches, hemorrhages or exudations could be detected in the eyeground.

O.S.—V. with $+1^{\circ}$ axis 90 was 6-5; the rotations of the eye-ball were normal in all directions; the fundus presented almost exactly the same appearances as those upon the opposite side; downward and inward there was a slight patch of super-

ficial choroidal disturbance. It will be seen, then, that the original oculo-motor palsy had subsided, although no single muscle, with the exception of the levator, had been restored to the normal.

The treatment has consisted of galvanism to the ocular muscles and ascending doses of strychnine. An obturator was placed over the right lens so as to neutralize the annoying crossed diplopia. In exactly one month there was slight improvement, so that the patient by a strong effort could overcome the divergence and rotate the eye about eight degrees towards the nasal side, while the upward rotation had become normal and the downward rotation had gained 10 degrees.

From the *résumé* given at the beginning of this paper it will be seen that this extraocular muscle palsy appeared at the time at which these paralyzes have been most commonly observed, namely, during convalescence. While we might naturally attribute the oculo-motor paralyzes to a meningitis, it would seem from the investigations of Osler that even in those cases in which all of the symptoms point to meningitis, where, for example, there are headache, photophobia, retraction of the neck, twitching of the muscles, rigidity and even convulsions, the actual pathological lesions of meningitis are not usually found. Thus, he made post-mortem examinations in three such cases, in two of which the diagnosis of cerebro-spinal fever had been made, and found no trace of meningeal inflammation, only congestion of the cerebral and spinal pia. Meningitis, however, does occur, but is uncommon, according to the Munich record, quoted by Osler, only 11 times among 2,000 cases. If meningitis is set aside as the etiological factor in cases of this character, they may be attributed to the poison acting directly upon the nervous system, affecting, for example, the oculo-motor, precisely as it may cause inflammation of other nerves, usually those of the arms and legs.

TRANSIENT PARALYSIS AS AN EPILEPTIC EQUIVALENT.¹

By J. W. McCONNELL, M.D.,

ASSOCIATE IN NEUROLOGY, PHILADELPHIA POLYCLINIC.

Transitory paralyses subsequent to epileptic seizures, whether such seizures are of the Jacksonian or ordinary type, are sufficiently well known to require little more than mention in this paper. Suffice it to say that they may be monoplegic or hemiplegic in variety, and may be due either to exhaustion or to inhibition. Other transitory paralyses related to epileptic attacks are of much less frequent occurrence, but at times occur, even some years previous to the onset of epileptic seizures; or, the epilepsy being established, they take place in the interval between the convulsions. Those which are observed before convulsions are seen at all, may be due to any one of a variety of causes; among which are syphilis, cerebral neoplasm, cerebral softening, general paralysis, etc. Of those occurring in the interval of epileptic convulsions, in illustration of which I here report a case, the cause seems to be, like that of the major condition, somewhat obscure.

H. is an intelligent unmarried female, 37 years of age, a native of the United States, and without any especial business. Her family history includes cases of degenerative mental disease, organic nervous disease, and tuberculosis; but no epilepsy, chorea or any form of convulsive disorder. She had convulsions when nine months old, and had them subsequently at intervals until three years of age. For four years she was free from seizures, but at seven years she had two convulsions in twelve months. Up to this time the attacks were mostly unilateral, involving the left side. At fourteen years of age menstruation started and the appearance of the menstrual flow was associated with general convulsion. From that time to the present she has had convulsive seizures at irregular intervals, although for about five years they have been greatly diminished in frequency and lessened in severity. The attacks are undoubtedly epileptic. She has never had any post-paroxysmal paralysis or paresis, or any abnormal mental condition. For the past two and one half years H. has had occasional attacks of paralysis involving the left arm and the left leg, either sep-

¹Read before the Phila. Neurological Society, April 24, 1899.

arately or together. The attacks last from a few seconds to two or three minutes; consciousness is never lost; they are preceded by prodromata in the form of paresthesia which involve the limbs to be paralyzed. When the paralysis is developed, whatever the patient is holding in the hand is dropped; or if she is standing and the leg is affected, she will sink to the floor. There is no aphasia, no amnesia, no involvement of the cranial nerves or of general sensibility. Control of the sphincters is perfect. After the spell is over the patient proceeds with whatever she was doing when it came on, and this occupation is sometimes not interrupted by the attack. The attacks occasionally take place at night, the patient being roused from sleep by the paresthesia. The paralyzes never come on during or after a convulsion, but rather in the interval between the convulsive seizures.

Summarizing, we have a person, the victim of epilepsy, who has at times paresthesia followed by paralysis, the loss of power being transient, unaccompanied by loss of consciousness or memory, or by any involvement of the cranial nerves. No convulsive movements either precede or immediately succeed these attacks.

In searching for some explanation of these paralytic phenomena, I am utterly unable to find any satisfactory theory either as to their nature or as to their cause. Very few such cases are reported, and those which I have found I have embodied in this report.

Higier,² of Warsaw, recently reported the case of a boy who had epilepsy since infancy, although at the time of the report he had been free from convulsions for some time. At five and one half years he again had epileptic seizures, and associated with them were momentary attacks of paresthesia with paralysis of the right leg. Sometimes the weakness extended to the trunk muscles and to the right arm. If standing, the boy would fall, but would not lose consciousness. The attacks lasted ten or twenty seconds, were frequently repeated and were not accompanied by aphasia or mental confusion. The paralysis occurred during sleep, the boy being waked by the paresthesia. There were no hysterical stigmata.

The same author later cites³ the case of a girl who had in-

² Higier, H., *Neurolog. Centralbl.*, 1897, No. 4, p. 152.

³ Higier, H., *Deutsche Zeitschr. f. Nervenheilk.*, V. 14, 1899, Nos. 3 and 4, p. 325.

flammation of the brain at three years of age, with convulsion and subsequent contracture of the left upper limb. In the last few months she has had in the intervals between epileptic seizures attacks of paralysis in the left lower and upper limbs, preceded by paresthesia, unaccompanied by loss of consciousness. The paroxysms last from ten to twenty-five seconds, during which time the left arm, usually contracted, is flaccid and its immobility disappears.

Pitres⁴ tells of a man who had epilepsy from childhood, and who at intervals during the course of that disease had transient paralyzes of the right upper limb, without preceding convulsion.

The case is recorded by Bouchaud⁵ of a woman fifty-three years of age who had repeated attacks of fugacious brachial monoplegia during fifteen years. Finally epileptic seizures of the Jacksonian variety supervened and were associated with paralysis in the epileptic interval.

Wood,⁶ years before the varieties of epileptic attacks were differentiated, claimed to have observed epileptic seizures which occurred "without loss of consciousness, with paralytic sensations and loss of power of particular limbs."

Whether or not these transient paralyzes can be classed as attacks of petit mal, or whether we can consider them as actually taking the place of the epileptic seizures, is a question. Petit mal consists merely in some transient loss of consciousness and very little or no convulsive movement,⁷ although occasionally we have only spasm without loss of consciousness.⁸ In the case here reported there has never been the slightest loss of consciousness in connection with the paralyzes, nor has there been the suggestion even of a spasm. This statement is based upon my personal observation of many of the attacks of paralysis, and upon the statements of the patient, who is a very intelligent woman despite her affliction. Certain it is, the

⁴ Pitres, A., *Revue de Médecine*, V. 8, 1888, p. 609.

⁵ Bouchaud, *Journal de Neurologie*, October 5, 1898, p. 392.

⁶ Wood, G. B., "Treatise on the Practice of Medicine," 5th ed., 1858, p. 785.

⁷ Hamilton, A. McL., "Syst. of Pract. Med.," ed. by Wm. Pepper, M.D., 1886, V. 5, p. 477.

⁸ Gowers, W. R., "Epilepsy and Other Chronic Convulsive Diseases," 1885, p. 90.

paralyses do not exactly resemble petit mal; they do not follow the convulsive seizures; nor, on the other hand, do they immediately precede them. They occupy a separate and distinct position, in which they appear to be the substitutes of the convulsive attacks, or, as some have called them, the "equivalents" of epilepsy. Pitres⁹ is one of the writers who has described these equivalents, and in commenting on them says: "It is less commonly known that epileptic patients may have without initiatory convulsion sudden transient paralyses, limited to a limb or extending throughout one side of the body. These transient paralyses appear suddenly in the midst of perfect health."

Higier¹⁰ regarded his two cases as instances of partial paralysis appearing as an epileptic equivalent, and employs arguments to support such a belief as follows:

1. In typical epilepsy the patient sometimes has attacks in which unconsciousness occurs, but no convulsion.

2. A brief paralytic period is observed after typical Jacksonian epileptic attacks, although the paralysis is not always proportionate to the convulsion. We also have partial epilepsy with simultaneous convulsion of one side and paralysis of the other.

3. Weakness of one limb may develop with, or precede, a Jacksonian convulsion.

4. In cases of partial sensory epilepsy loss of function of a sensory organ occasionally takes place, and sometimes rare cases of both motor and sensory inhibition are seen.

5. Ophthalmoplegic migraine is an undoubted epileptic equivalent, and here loss of function (ocular paralysis) is prominent.

6. Chorea has many clinical and pathological similarities to epilepsy, and of this a paralytic form¹¹ (chorea mollis s. paralytica) is seen, in which paralysis of one or both sides precedes the clonic stage.

While Higier's arguments are intended to prove the occurrence of these motor paralytic equivalents, it seems to me he rather relates them to post-paroxysmal paralyses, or, at least,

⁹ Pitres, *op. cit.*

¹⁰ Higier, *op. cit.*

¹¹ Gowers, W. R., Brit. Med. Jour., April 23, 1881, p. 636.

his distinction between the two is not very clear. His object is evidently to show the frequency of the occurrence of paralysis in epilepsy and allied diseases.

In studying the subject of transient paralyses one symptom group, recently so well studied by Taylor,¹² has impressed me as perhaps in some way related to these transient paralyses occurring in the epileptic intervals. This is the so-called family periodic paralysis. I do not assert that there is an absolute relationship between the two conditions, but some points of resemblance suggest such a possibility. These points of similarity are the influence of heredity; the recurrent character of the conditions; the occurrence of prodromata; the frequent nocturnal onset of the troubles; the retention of consciousness throughout the duration of the paralysis; the headache or slight indisposition following the attacks. The peculiar electrical changes in the muscles in cases of family periodic paralysis have not been observed in the transient paralyses of which my case is an example, nor are diminished reflexes noted. Both conditions are paroxysmal appearances of limited disturbance of the motor spheres, although of different duration. In either the loss of function may be a monoplegia,^{13 14} or a hemiparesis,¹⁵ although usually in family periodic paralysis both lower extremities are involved. Mellus,¹⁶ in the discussion of the case of family periodic paralysis presented before this society by Dr. John K. Mitchell some months ago, said that the history of the patient suggested to him some of the cases of post-paroxysmal paresis without epileptic paroxysms.

In looking for the cause of these transient paralyses occurring in the epileptic interval I find little to throw light upon the subject, or to explain how transient paralyses can replace or can substitute epileptic convulsions.

Grasset¹⁷ considers the two conditions, paralysis and con-

¹² Taylor, E. W., Jour. Nerv. and Ment. Dis., V. 25, 1898, Nos. 9 and 10, pp. 637, 719.

¹³ Bouchaud, *op. cit.*

¹⁴ Hirsch, K., Deutsche med. Wochenschr., 1894, No. 32, p. 646.

¹⁵ Burr, C. W., Univ. Med. Mag., Phila., 1892-3, p. 836.

¹⁶ Mellus, E. L., Jour. Nerv. and Ment. Dis., 1899, Vol. 26, No. 3, p. 178.

¹⁷ Grasset, J., et Rauzier, G., "Traite pratique des Maladies du Système nerveux," 4th ed., 1894, V. 1, p. 284.

vulsion, quite independent of each other, hence, says Pitres,¹⁸ "it is possible that they, the paralyzes, are dependent upon a sudden and temporary impairment of the motor centers." But what is the cause of this sudden impairment? Jackson¹⁹ says that repeated transient paralyzes of similar local involvement are hard to explain, but he offers the suggestion of a small clot or brain softening from occlusion of a small vessel. Bouchard²⁰ ascribed the symptoms in his case to a deep-seated cerebral glioma, although the presence of a neoplasm is not well made out in the clinical history of his patient.

The agency of auto-intoxication in the production of these motor paralytic equivalents is a theory not to be entirely set aside. Transitory paralyzes, not necessarily related to epilepsy, sometimes occur as the result of toxemia. Achard and Levi²¹ have reported cases of transitory paralyzes of cardiac origin and relate them to those due to uremic intoxication and to the blood poisoning from hepatic disease. Van Gieson²² believes that by the action of a toxic substance upon the lowly organized neuroglia cells and upon the mesoblastic connective tissue cells, increase of the neuroglia cells in the subcortical zones can readily be produced. Such a pathologic finding he has made in the brains of epileptics. Von Jaksch²³ found that epileptic attacks may be caused by auto-intoxication from urea or acetone, without the presence in the urine of sugar or albumin. Mairret²⁴ found that before an epileptic seizure the urine was hypertoxic, and that subsequently and in the interval it was hypotoxic. Cabitto²⁵ employed the sweat secreted in the prodromal and early stages of an epileptic convulsion and produced in rabbits similar convulsive conditions. No such result was obtained from the secretion of the decreasing stage or of the interval.

Making comparison again with family periodic paralysis, we find that Taylor²⁶ thinks that auto-intoxication is "by all

¹⁸ Pitres, *op. cit.*

¹⁹ Jackson, J. Hughlings, *Brain*, Jan., 1881, V. 3, p. 437.

²⁰ Bouchard, *op. cit.*

²¹ Achard and Levi, *La Méd. moderne*, 1897, 7, p. 656.

²² Van Gieson, *Ira*, *State Hosp. Bull.*, N. Y., 1896, V. 1, No. 4, pp. 470, 471, 472.

²³ Von Jaksch, R., *Zeitschr. f. klin. Med.*, 1886, p. 199.

²⁴ Mairret, *La France méd.*, 1897, No. 5, p. 70.

²⁵ Cabitto, C., *Riv. Speriment. di Freniat.*, 1893, 27, p. 36. Abstract in *Jour. Nerv. and Ment. Dis.*, 1898, V. 25.

²⁶ Taylor, *op. cit.*

means the most plausible working hypothesis" in seeking a cause for this symptom group, "the source of origin of the problematic poison being wholly obscure." This opinion is shared by Mills²⁷. Goldflam²⁸ found the urine of a patient suffering with family periodic paralysis to be hypertoxic during the attack, and hypotoxic after recovery and in the interval. If auto-intoxication can cause, or be supposed to cause, transient paralyzes of the family type, the origin of somewhat analogous loss of power might be considered possible of similar explanation, until at least, we know more of the exciting causes of epilepsy.

²⁷ Mills, C. K., Jour. Nerv. and Ment. Dis., 1899, V. 25, No. 3, p. 178.

²⁸ Goldflam, quoted by Van Gieson.

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107. ZUR PATHOGENESE DER HEMICHOREA POSTAPOPLECTICA (Contribution to the Pathogenesis of Post-apoplectic Hemichorea). W. Muratow (Monatsschrift für Psychiatrie und Neurologie, Vol. 5 1899, p. 180).

Muratow reports two cases, one with necropsy, as a contribution to our knowledge of forced movements. They seem to show that a lesion of the fibers forming the anterior cerebellar peduncle and the nucleus ruber causes hemichorea or athetosis. He believes that Bonhoeffer has definitely proven that a lesion of these fibers produces such movements. Muratow's second case is evidence that hemichorea may result from a primary lesion of a cerebellar hemisphere and secondary degeneration of the anterior cerebellar peduncle. He believes that post-apoplectic forced movements indicate a disturbance of co-ordination in the functions of the cerebellum, basal ganglia and the cells of the anterior horns. Involvement of the cerebral cortex and of the pyramidal fibers cannot be excluded, but the cortex is involved only by means of the afferent tracts, and focal cortical lesions of the central convolutions do not produce such involuntary movements. He speaks, however, of certain forms of cortical forced movements which he does not separate sufficiently sharply from the sub-cortical forced movements. Athetoid and choreic forced movements are not associated with disturbance of the muscle-sense or cortical epilepsy. Muratow finds that a lesion of the nucleus ruber causes forced movements on the opposite side of the body; and one of the cerebellum, forced movements on the same side. Post hemiplegic tremor is a spinal symptom, and indicates an increased tonicity of the cells of the anterior horns.

SPILLER.

A CASE OF PURE PSYCHICAL EPILEPSY.

By JOHN NORMAN HENRY, M. D.,

VISITING PHYSICIAN TO THE CHURCH HOME FOR CHILDREN, ANGORA, PA.;
PHYSICIAN TO DISPENSARY ST. CHRISTOPHER'S HOSPITAL, PHILADELPHIA.

The man whose case I am about to report first came under my notice at the Pennsylvania Hospital in the wards of Dr. Morris J. Lewis, through whose kindness I am enabled to make the following excerpts from the hospital notes:

W. E., aged forty-three years, widower; occupation, general business; was admitted on April 20, 1897.

Family history: Mother died of Bright's disease. Father living. Family history was otherwise negative.

Previous history: Has had several attacks of gastritis, and many years ago had an attack of rheumatism. Used to drink to excess; now does not drink at all, and has not for three years. No venereal history. Three years ago he had hemorrhages from mouth and gums. Is positive that the blood did not come from lungs or stomach. Previous to this his legs had been slightly swollen. He was treated in a New York hospital and recovered after a stay of several weeks.

Present illness: On admission he complained of swelling of legs and abdomen, also of shortness of breath on exertion. Has recently had slight hemorrhages from the gums. Has no disturbance of the stomach; has not been jaundiced. Has been passing from 35 to 50 ounces of urine *per diem* for at least two weeks, according to his own measurements.

Physical examination: Poorly nourished and sallow looking. Tongue dry and flabby; pulse accelerated, regular; arteries hard; pupils normal; abdomen very much swollen; legs swollen and edematous; scrotum also much swollen. Veins in abdominal wall markedly enlarged. Liver impossible to outline on account of the swelling of the abdomen, but is apparently enlarged. Spleen, normal in size, apparently. Heart: at apex there is a presystolic and systolic murmur, while over the aortic cartilage is also heard a systolic murmur transmitted into the vessels of the neck. Lungs: there is dulness at both bases, with absence of breath sounds. There is superficial edema over the back generally. Urine: amber in color; acid in reaction; sp. g., 1.018; contains no sugar or albumin; a few fine hyaline and granular casts. Treatment: Basham's mixture f. $\frac{3}{4}$ ss. t. d.

April 25. Has some difficulty in sleeping and complains of nervousness; edema of legs and abdomen decreasing.

April 28. Liver can now be palpated and is enlarged slightly.

May 3. Doing well. Does not sleep much at night and is very nervous.

May 13. Still very nervous. Is able to be up for a short time daily. Urine examination negative.

From the time of this note until the 24th of June, on which date he was discharged from the hospital, he steadily improved and left practically well. From time to time he used to report himself to me at the hospital, and except for some slight edema of the feet at night after an unusually active day's work, he seemed to be in excellent health.

On May 4, 1898, he consulted me in my office, complaining of weakness, nervousness, shortness of breath on exertion, and some slight edema of the feet toward evening. Examination of heart showed some hypertrophy, associated with systolic mitral and aortic murmurs. Heart sounds were regular and of good force. Examination of urine showed a very slight trace of albumin, a specific gravity of 1.018, and a very few fine, pale granular casts. The liver was a little enlarged, being about one finger's breadth below the costal margin. He was ordered tincture of digitalis, gtt. xv., morning and evening, and strontium lactate, and was advised to lie down for one hour in the middle of the day. He seemed to steadily improve in general condition, and in ten days the urine examined showed no albumin and no casts.

On May 15, 1898, he came to my office in a very nervous state, and gave me the following account of himself:

At 3 P. M. on the preceding afternoon, while sitting in his office, he began to feel stupid and heavy, and acted in a manner that aroused comment and questions from his father and cousin, who were present, at which he showed great irritability. He also remembers feeling nauseated, but did not vomit.

At 6 P. M. his cousin took him to within two blocks of his home and left him, after being assured that he (the patient) could easily take care of himself for the rest of the way. Up to this point his consciousness was present, though evidently, from his own description, somewhat benumbed. His next recollection was finding himself at 2 A. M. on the following morning in an open lot in an outskirt of the city, trying to climb over a fence in order to board a passing trolley car, which he succeeded in doing. He states that he felt dazed and surprised at finding his whereabouts and had no recollection of intervening events. He again relapsed into unconsciousness, was carried by the car far beyond his destination, and awoke to find himself walking on a street in another outskirt of the city almost as much below his house as the point of boarding the car had been above it. For a second time he took a car, was again carried beyond where he should have gotten out, and found

himself walking aimlessly along a street about two miles from his home, from which place he walked, fearing to trust himself again to the car, and arrived safely at home utterly exhausted and profoundly sleepy.

There was nothing unusual in his condition when he came to my office that morning. He had no headache, his mind seemed perfectly clear, urine was passed freely, and contained neither albumin nor casts. He denied having taken any alcohol. I advised him to carry a small bottle of aromatic spirits of ammonia, and to take a dose if he should again feel an attack of the same character coming on; also to let his family know immediately of his condition.

On May 26, 1898, he was brought to my office by two of his friends just as he was becoming conscious, after having had a second attack of a similar nature to the one before described. I was, unfortunately, out at the time, but my father, Dr. F. P. Henry, happened to meet him on the doorstep, and was able to satisfy himself and me that there was no question of drunkenness in the case. On the following morning he gave me these details of his attack:

He began in the afternoon to feel dull and stupid, was again nauseated as in the previous instance. He was able, however, to dress and go out for dinner. He found the right house, but was discovered trying to open the door with his own key. Concerning the events of the rest of the evening his memory was rather vague, though he did remember being laughed at for attempting to eat soup with a fork. He became conscious or semi-conscious to find himself lying on a sofa, probably about two hours after the dinner hour, and asked to be taken to my office; and though he could distinctly remember and articulate clearly my name, he was entirely unable to tell on what street I lived. This difficulty, however, was overcome by his directing his friends to the proper car and by his recognizing the proper street at which to get off. By the time he had reached my house his mind had cleared up, and except for some unsteadiness of gait he appeared to be quite himself again. He slept soundly all night, and in the morning felt well. He was subsequently informed by his friends that he was able to converse during any time of his attack, but was very irritable and disagreeable. He had no convulsion.

After this second attack I inquired more carefully into his family and personal history, having the suspicion of epilepsy in mind, but found the family history perfectly free from any nervous taint whatever. In the matter of his own history, he had never had either grand mal or petit mal, and was able to tell of nothing which could suggest nocturnal attacks.

On June 1, 1898, he had his last and most serious attack.

On Friday afternoon, June 3, I was called to his house, and was somewhat surprised to learn that he had spent the last few days in the insane department of the Philadelphia Hospital. His story is much as follows: On the morning of June 1, while at his office, he began to feel dull, stupid, and unable to attend to his business. About noon he left his office and went to his tailor's shop near by. After leaving this place he is able to remember nothing until 9 A. M. on the following morning, when he awoke to find himself strapped in bed at the Philadelphia Hospital in the insane wards. He had lost his watch, chain and a scarf-pin during his wanderings, and on placing the matter in the hands of the police he was able to follow up his own trail to a certain extent. He learned that he had been arrested near the City Hall at about 1 A. M., after attempting to get into a car through the front window by standing on the fender. He had previously been seen to kick over some bicycles and deliberately stamp on them; also to try to enter a large store through the window. He was taken to the City Hall, where he was recognized and questioned, but to all inquiries he gave unmeaning answers and showed great irritability. He was then seen by a physician, and sent to the Philadelphia Hospital in a patrol wagon after vain attempts to learn from him the address of his father, who had recently moved, and whose address consequently did not appear in the city directory.

I saw the resident physician at the Philadelphia Hospital under whose care my patient had been while in the hospital. The doctor told me that Mr. E. on his arrival was very much excited, and was so violent that it was necessary to confine his hands and feet to the bed with straps; that he showed no paralytic or convulsive symptoms; his pupils were equal and normal in size. He had also a curious aphasia, in that he had but two words in his vocabulary, one being his own name, the other the word "no," either of which he would answer indiscriminately to all questions put to him. There were no signs of alcoholism about him. During the night he evacuated his bowels and bladder in bed. By 9 A. M. on June 2 he had become conscious and rational; the aphasia had disappeared, and he was able to walk about the ward, and on the following day he was discharged.

As in the other instances following this attack, there seemed to be nothing unusual in his condition. His mind returned to its normal functional activity; he had no headache or dizziness, and he again appeared to be in good health, except for the natural anxiety which he now began to feel in regard to the possibility of frequent returns of these attacks, which to

some degree influenced his general health. His eye-grounds were examined and the fields of vision taken, and both found to be normal.

He was immediately placed on bromide of potassium 10 grains, and iodide of potassium, 5 grains, three times daily, but as he showed great susceptibility to the iodide it became necessary to reduce the dose of it to one grain morning and evening, continuing the bromide as originally prescribed. From that date, *i. e.*, June 3, 1898, until January 3, 1899, since which time I have lost track of him, he had no further attacks. The case is one of considerable interest from various standpoints.

Briefly stated, the points in regard to the diagnosis are these: A man, forty-three years of age, with a history of great alcoholic excesses extending up to a period of time within three years of his coming under observation, free, however, from any family or personal history of epilepsy in any of its phenomena, has a series of three attacks of psychical aberration lasting for several hours, each similar in onset, symptoms, actions and subsequent course; while during the intervals between these attacks he enjoys perfect mental health, associated with as good physical condition as is compatible with the condition of his heart, kidneys and liver.

These attacks were in each instance preceded by a feeling of dulness and mental hebetude, followed by nausea without vomiting; upon this condition of dulness rapidly ensued that of unconsciousness, lasting for several hours, during which automatic actions were performed, conversations held and distances traversed in cars (the car-fare presumably being paid also). On the administration of bromide and iodide the attacks disappeared.

There is also another point which in the recital of his story I failed to bring out; namely, that during the unconscious periods consciousness would recur to him again and again for a few seconds, in which he would notice the name of a street, recognize a shop, or receive the impression of a face which he may still remember. Without overstraining the analogy to motor epilepsy we might compare the periods of unconsciousness with the waves of consciousness to the tonic and clonic phases of a motor spasm.

The age of the man, the absence of epileptic taint in the

family, and also of any other epileptic manifestations in the man himself, made me slow to accept the diagnosis; while on the other hand, the history of alcoholic excess, so potent an etiological factor in epilepsy in later life, the presence of an aura, represented by the nausea which preceded each attack, the perfect mental equilibrium which intervened, the absence of any signs of organic brain disease, and the prompt and lasting response to the administration of bromide, logically justifies the diagnosis of pure psychical epilepsy—"épilepsie larvée pure" of the French.

Much has been said and written on both sides of the question in regard to the existence of a pure psychical equivalent of epilepsy, most of the English writers taking the view expressed by Savage and supported by Hughlings-Jackson, that a fit of some kind, great or small, must precede the unconsciousness. While there can be no doubt that this is almost the invariable rule, yet there are exceptions, and a pure psychical epilepsy, however rare it may be, is fairly exemplified in this case.

All three of his attacks occurred in the daytime in a populous part of a large city; he was under observation of friends at and before the beginning of each attack, during the attack and after the attack, and in one instance almost throughout the attack, and nothing corresponding to a fit, great or small, was observed.

The French writers accept the existence of pure psychical epilepsy. Ardin-Delteil in his recent work speaks most positively on the subject. He states that not only does the psychical equivalent occur in conjunction with grand mal, petit mal, Jacksonian epilepsy, as pointed out by Pitres, but also that it occurs entirely free from any other association. He considers the addition of motor phenomena in a case of psychical epilepsy as evidence of onward progress of the disease. He speaks of a case of a girl who had three attacks of unconsciousness during one week, in whom the motor symptom preceding the unconsciousness was confined merely to a twitching of the middle finger of the right hand. That motor epilepsy may be limited to any degree, even, as in the case just mentioned, to the center for a single finger, with or without unconsciousness following,

is a recognized fact. Recently also the writer has had knowledge of a case presenting a complete epileptiform seizure without any loss of consciousness whatever. In view of these examples of limitation of convulsion, it would seem reasonable to accept the possibility of a discharge entirely limited to the intellectual area.

Fortunately this case had no legal bearing, though it is evident from the history that the patient would have been quite capable of carrying out any violent impulsive idea had the suggestion occurred to him, and that he would have been entirely irresponsible in the matter.

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108. ZUR LEHRE VON DEN GESCHWÜLSTEN DER RÜCKENMARKSHÄUTE (A Contribution to the Study of Tumors of the Spinal Membranes). A. Fraenkel (Deutsche med. Wochenschrift, 1898, 28, 29 and 30, pp. 442, 457 and 476).

Fraenkel reports two cases of tumor of the spinal membranes. In the first case a gliosarcoma surrounded the cervical portion of the spinal cord like a mantle, 3 to 4 mm. thick. It was less extensive in the thoracic region, but the cord at about the fifth thoracic segment was involved in the growth. Numerous nodules were found on the nerve roots in the lumbar region and on the cauda equina. The disease began with pain in the muscles of the neck and back, and this was followed soon by the signs of a transverse lesion of the cervical cord. The duration of the process was only about six weeks. The diagnosis of tumor could not be made during life, as the symptoms indicated a myelitis of the cervical swelling.

In the second case the cord was examined only below the cervical region. A tumor, of about twelve cm. long in the thoracic region, and nodules lower in the cord, were found. At the lower thoracic region the cord was involved in the growth, and here numerous canals and spaces were found lined with a single layer of cylindrical cells, resembling the epithelium of the central canal. Fraenkel believes that these cell nests were probably separated from the tissue about the central canal early in life, or even in the embryonic period, and after an injury had been received, gave rise to the growth. Only in the portion where the cord was involved did the tumor present the appearance of a neuroepithelioma (Rosenthal); elsewhere it was a gliosarcoma without cell-lined spaces.

He states that sarcomata involve chiefly the posterior part of the pia, and thereby cause great pain, which, however, is not a pathognomonic symptom, and in some cases is not pronounced. He calls attention to the slight tendency to involvement of the cord and nerve roots manifested by the tumors in his two cases.

Benda examined the tumor in the second case and makes some interesting remarks on the origin of the glia and ependyma. He believes also that the growth began in the epithelial nests separated from the central canal, but as ganglion cells were not found in the tumor, he prefers to call it an ependymoma instead of a neuroepithelioma, in order to avoid confusion. He recommends a special method of staining.

SPILLER.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

April 4, 1899.

The President, Dr. Frederick Peterson, in the chair.

ALCOHOLIC NEURITIS IN A CHILD.

Dr. George W. Jacoby presented a boy, four years and a half old, exhibiting symptoms of alcoholic paralysis. When first seen on February 20, it was stated that the boy had been well up to four weeks previously, at which time he had had severe colic without vomiting or constipation. Then the left knee-joint had become swollen. Eight days previously he had been noticed to be unsteady in walking. It was found that the boy had received about a half to one tumblerful of beer daily ever since the age of six months. Examination had revealed extensor paralysis of the hands and legs, with a reaction of degeneration in all the muscles. The extensors of the thighs were unaffected, and there was no sensory disturbance. The speaker quoted some recent statistics regarding the habitual use of alcohol in a large German city. The municipal authorities had undertaken an investigation among the school children, and had found that of 100 children, 16 drank no milk. Twenty-five per cent. of the children had never tasted brandy, but had habitually drunk beer or wine. Eight per cent. had received their daily portion of brandy "to make them strong." He thought there was an almost equally large percentage of children among the German and Irish population here who were habitually given alcoholic drinks. This case emphasized the cumulative effect of small doses of a poison long continued.

Dr. William M. Leszynsky agreed thoroughly with Dr. Jacoby regarding the prevalence of this vicious habit in this country.

Dr. Joseph Collins said that he had two such cases under observation in the last two years. One of these, a child of seven years, was now passing through the second attack. This child had been in the habit of drinking beer. He had not completely recovered his muscular power when the second attack came on. Dr. Collins had been impressed with the phenomenon presented by all the cases that he had seen, and which was present in the case just exhibited, *i. e.*, the remarkable pallor of the cutaneous surface as compared with the redness of the mucous membranes. In this connection it was interesting to note that Hughlings Jackson had recently recorded himself again in favor of treating chorea entirely by the use of port wine.

Dr. Frederick Peterson remarked that it was very unusual for an alcoholic neuritis to be free from sensory symptoms.

Dr. Jacoby said that there was intense pain over the nerve trunks in his case, but there was no general hyperesthesia of the skin.

AN EPILEPTIC PATIENT WITHOUT AN ATTACK FOR FOUR YEARS.

Dr. J. Arthur Booth presented a woman, fifty-one years of age, as a case of cured epilepsy. The interesting point was that she had gone four years without an attack. She had been under his care since 1882. She had enjoyed good health up to the twelfth year, when she had had her first seizure, characterized by loss of consciousness, frothing at the mouth, biting the tongue and deep stupor. From this time up to 1880 there had been about six attacks each year. For the next two years the seizures were more frequent and severe, so that they occurred several times a week. The attacks were apparently not influenced by menstruation. She had been given bromide of sodium and chloral. At one time she had received as much as four drams of the bromide in the twenty-four hours. During the past year she had only taken ten grains daily.

Dr. Peterson remarked that in view of the investigations of Dr. Sinkler it did not seem proper to call any case of epilepsy cured, although ordinarily a case that had gone as long as four years without an attack might be considered as cured. A collective investigation on this subject would prove very instructive. Probably a much larger percentage was cured by drugs than by surgical operation.

Dr. Edward D. Fisher said that children often had attacks for several years, and then there was an interval of four years or more before their return. He had known cases to pursue such a course with little or no treatment, and hence it was not even presumptive evidence of a cure. He had not found anything to take the place of the bromides, and considered this the proper treatment. In his experience the continuous use of the bromides had not had any deleterious influence on the mind.

Dr. George W. Jacoby said that one must place some limit to determine what should be called a cure. An epileptic patient might have a return of his trouble just as he might of other disorders. An interval of many years followed by a return of the seizures he would rather look upon as a fresh attack of epilepsy brought about quite possibly by the same cause as had originally produced it.

Dr. L. Pierce Clark said that he had known several cases of epilepsy to apparently undergo a spontaneous cure, that is, they would have no seizure for perhaps thirty years, and this, too, without any apparent relation to medicinal or dietetic treatment.

Dr. Booth said that the result of drug treatment in epilepsy depended very largely upon the faithfulness with which the treatment was kept up, and it was because patients were usually negligent that more so-called cures were not reported.

TWO CASES OF LOCALIZED SCLERODERMA.

Dr. George W. Jacoby presented two women showing localized scleroderma. Both upper extremities and the face

were affected in both persons. One patient, a girl of seventeen years, had been quite sickly in infancy. At the age of two years she had been scalded from the shoulders to the waist. At the age of five years stiffness of the hands had been first noticed. The other patient was thirty-five years of age, and had been a healthy child. She had been married at the age of twenty-two years, had had six healthy children, and had herself enjoyed good health. Seven years ago she had noticed a sore on the tip of the index finger of the left hand, and this had healed very slowly. Then the other fingers became the seat of open sores, and as they healed the fingers were noticed to be stiff. On examination, both patients presented hardness, stiffness and bronzing of the skin, together with wasting. There was an absence of the first phalanx of each finger. These cases were presented because of their comparative rarity. He had only seen three other cases. In two of the latter there had also been a history of scalding or of severe injury of some kind to the skin. He was inclined to think that these cases might not be tropho-neuroses, as was generally believed, but instead examples of parasitic infection. Both of the patients presented had been under treatment with the desiccated thyroid, one of them sufficiently long to show that the treatment was in her case an utter failure. The question always arose as to the possibility of this affection being dependent upon some interference with the function of the hypophysis.

Dr. B. Sachs said that about two months ago he had seen a gentleman who had fallen off a ladder, and in doing so one hand had been penetrated by a sharp steel wire. Soon after this accident there had been flushings connected with a part of the distribution of the musculo-spiral nerve. Subsequently a very marked and typical scleroderma had developed. The man had been under treatment with the thyroid, and was taking at the present time fifteen grains, three times daily. Under this medication the scleroderma had been distinctly diminished.

Dr. L. Stieglitz said that he believed he had been the first one in New York city to use the thyroid treatment in cases of scleroderma, and he had found that it was very beneficial during the hypertrophic stage. It was not surprising that in the stage of atrophy the method should prove unavailing. He had seen good results from this treatment in three cases.

Dr. Jacoby thought that localized scleroderma was different from the other forms of scleroderma—it was a distinct entity.

PROGRESSIVE MUSCULAR ATROPHY (?).

Dr. L. Stieglitz presented a man, sixty-two years of age, who six years ago had first noticed a weakness in the left leg. This had increased steadily, and had been associated with wasting. Two years ago he had been injured in a runaway accident, and asserted that he had been unconscious for a week. The pupils reacted to light and during accommodation. There

was perfect control of the sphincters. Examination showed marked weakness and wasting of the left thigh and calf, and beginning wasting on the other side. The knee-jerk was absent on the right side, and on the left there was only a response in the sartorius muscle. The man also had a dislocation upward of the left hip. The case suggested three diagnoses. The first of these was tabes with a Charcot joint in the left hip, but it was evident that there was no disturbance in the sensory part of the arc, and there was no other symptom of tabes. The second possible diagnosis was dislocation of the hip with secondary wasting of the muscles, but, according to the history, the atrophy of the muscles had begun long before the injury to the hip. The third diagnosis, and the one to which he inclined, was progressive muscular atrophy with incidental dislocation of the hip.

Dr. L. Muskens said that in this case he had found a lack of faradic response, more marked on the left side. As a result of his examination of the patient he took the same position as Dr. Stieglitz.

Dr. Joseph Collins said that the case had been under treatment at his clinic for a long time, and there had then been no suspicion of the existence of progressive muscular atrophy. In the hospital in which he had been previously there had been no history given of muscular atrophy prior to the injury to the hip. He looked upon the case as one of dislocation occurring in a man of very feeble recuperative power. The atrophy seemed to be entirely one of inactivity.

Dr. Leszynsky thought that this patient presented remarkable resistance power in his extensors, and the condition of the knee-jerk seemed to him to point to tabes.

Dr. Fraenkel said that the fact that the knee-jerks were lost on both sides would indicate a more serious condition than an inactivity atrophy. The resistance of the extensors could be explained by contracture of the muscles as a result of the injury to the hip.

Dr. Stieglitz opposed Dr. Collins' diagnosis as not being in accordance with the conditions found in the two lower extremities. Gowers had stated that the knee-jerk was often lost before there was marked atrophy in cases of progressive muscular atrophy. The response from the sartorius seemed to indicate positively that part of the sensory arc must be free.

DEMONSTRATION OF SPECIMENS FROM A CASE OF ERYTHROMELALGIA.

Dr. B. Sachs and Dr. A. Wiener gave this demonstration. Dr. Sachs said that this disease had been carefully studied in the past six or seven years. The only autopsy on record was on a case under the care of Auerbach, and it was associated with tabes. There were changes found in the lumbar and sacral roots, but it was not shown that the erythromelalgia was dependent upon central disease. Mitchell and Spiller had recently observed a case in which it had seemed to them that the nerves were more involved than the blood vessels. The speci-

mens demonstrated had been taken from a man, thirty-six years of age, who had been first seen in 1898. At that time, when the part was pendent a few minutes, there was a violet color of the foot and ankle, associated with severe pain and tenderness. A few months later a gangrenous ulcer formed on the dorsum of the foot. There was marked atrophy of the anterior tibial group. Owing to the rapid spread of the gangrene, it had been considered necessary to amputate through the thigh. According to Weir Mitchell there was no gangrene in cases of erythromelalgia, and the disease was asymmetrical, but in this case the appearance had been typical before the occurrence of gangrene, and moreover slight gangrene had been reported by others. Marked arterial changes were found in this case, particularly in the larger branches of the popliteal artery. This in itself seemed to be a sufficient justification for Dr. Gerster's opinion that the amputation should be done above the knee. The symptoms seemed to be explained by obliterating endarteritis. The changes in the nerves were so slight that he looked upon them as secondary. It was interesting to note in this connection that several cases of cardiac disease and marked arterial sclerosis were at the Montefiore Home presenting the principal symptoms of erythromelalgia. There was an interesting analogy between erythromelalgia and Erb's description of intermittent claudication.

Dr. A. Wiener said that specimens of nerve, skin, muscle and connective tissue had been given to him for examination, and in not a single one were the arteries normal. Only in the distal portions of the peripheral nerves could any distinct changes be found, and they were rather degenerative than inflammatory. In all of the specimens a very prominent feature had been the enormous quantity of connective tissue. He had not been able to find a single case on record in which arterial changes had not been found, whereas cases had been reported in which there had been no changes in the nerves.

Dr. Ira Van Gieson thought that in this case the vessels were affected to a somewhat greater extent than in the Mitchell-Spiller case. The specimens showed plainly the predominance of the arterial changes.

Dr. C. L. Dana said that this case differed essentially from the typical ones described by Weir Mitchell in the occurrence of gangrene, and hence it was possible that the histological appearances were not exactly those found in the classical cases of erythromelalgia. Two cases of erythromelalgia were cited in which the urine had contained sugar, and the patients had improved greatly under appropriate dietetic treatment. This seemed to suggest that there might be an underlying diabetic or gouty state leading to neuritis or disease of the blood vessels.

Dr. William H. Thomson spoke of cases of pseudo-erythromelalgia occurring in Graves' disease, and in allied cases characterized by persistent tachycardia. He had seen at least ten of these cases, and felt sure that when severe they might be very readily confounded with true erythromelalgia.

Dr. Leszynsky remarked that two years ago he had seen a case of acromegaly in which erythromelalgia had attacked the upper extremities.

Dr. Stieglitz spoke of a case of typical erythromelalgia which he had seen about two years ago in a drug clerk. After a few months attacks of local asphyxia, associated with severe pain, had developed in the toes. Under rest in bed and the use of the iodides the improvement had been decided. Aside from the features mentioned all the classical symptoms described by Weir Mitchell had been present, yet if the case had been allowed to go on unchecked he was confident that gangrene would have developed.

Dr. Sachs, in closing, insisted that the case had been a typical erythromelalgia for several months, and that the gangrene had developed at a very late stage. The fact that it occurred in a man of thirty-five years, apparently healthy in every other way, was very interesting. The man had recovered very satisfactorily from the amputation, and the pain had entirely disappeared.

REMARKS ON TWO CASES OF BRAIN TUMOR, WITH PRESENTATION OF SPECIMENS.

Dr. Joseph Collins reported these cases. The first was that of a child of six and a half years. When four years old there had been an attack of measles, followed by slight otorrhea on one side. The last illness had been ushered in by severe headache and persistent vomiting, and the latter had lasted for about three months. Relief had been sought chiefly because of the intense pain and the trembling in the hands. Examination showed that the patient did not use the left hand as freely as the right. There was no control over urination and defecation. There was double choked disk. The diagnosis of cerebellar tumor, probably vermicular, was made. The strabismus which was present was not constant. The temperature was elevated from one to five degrees during the last few days. The autopsy revealed a lobulated mass, the size of an English walnut, projecting from between the hemispheres of the cerebellum. It had caused atrophy of portions of the right hemisphere of the cerebellum. The gross appearance of the tumor was that of sarcoma.

The second case was that of a woman, twenty years of age, who had been in good health up to November, 1897, or about seven months before coming under his observation. The initial symptom—intense pain all over the head—had appeared very abruptly, and had been associated with vomiting and dimness of vision and progressive dementia. The left eye showed distinct choked disk; the right eye great swelling of the veins. Three days after admission to the hospital an attack of syncope occurred. She was six or seven months pregnant. One month later she became restless and irritable, and developed fever. Labor was induced, but the patient died the next day.

The autopsy revealed a sarcoma the size of a hen's egg in the anterior third of the right lateral ventricle. Both ventricles were distended with fluid.

TEMPORARY (EXHAUSTIVE) PARALYSIS IN EPILEPSY.

Dr. L. Pierce Clark presented a brief abstract of his paper on this subject. An analysis of the cases therein reported was as follows: Cases of local exhaustion with general seizures; cases of paralysis at the beginning, and becoming, to a certain extent, permanent; exhaustion paralysis in infantile cerebral palsy; exhaustion paralysis associated independently with cerebral palsy, but on the opposite side to the organic lesion; cases of exhaustion manifested in aphasia only; cases of paralysis manifested in the right leg and left arm. His conclusions were: That the theory of exhaustion paralysis had been conclusively proved by physiological experimentation, and that exhaustion paralysis, generally localized to the parts participating in the spasm, was confined to those parts most convulsed in general seizures. There might be exceptions to this general rule. The transient paralyzes might, after a time, become permanent hemiplegias. Temporary exhaustion paralysis was essentially exhaustion of cerebral centers, and the frequency or severity of the muscular spasm was not a fair index of the amount of exhaustion of these centers. A careful study of exhaustion phenomena suggested that epilepsy was clearly allied to paralytic states, due allowance being made for the fact that the epileptic brain did not possess the normal capacity. The great frequency of epilepsy as a symptom following most cerebral lesions of a transient nature tended to substantiate the clinical hypothesis of the close association of epilepsy and paralysis. Minute disseminated patches of sclerosis in different areas of the epileptic brain had been repeatedly demonstrated by various observers, and this also helped the hypothesis very strikingly. In not a few instances he had produced apparent epileptic seizures by massage.

Dr. Dana said that the photographs presented in connection with this paper gave a more graphic description of the epileptic convulsion than any others he had seen. They should prove very helpful to teachers. He thought when neurologists were able to make a closer study of the clinical phenomena of epileptic seizures they would be in a better position to localize the disorder.

Dr. I. Van Gieson said that the theories of epilepsy were not thor-

oughly satisfactory, and one of the advantages of a place like the Craig Colony was the opportunity afforded for close and prolonged study. He predicted that in these cases evidence of an abnormal expenditure of energy in the cortical motor cells would be found. In one case that he had studied, that of a patient who had been in the City Hospital, and had had sixty seizures in one day, unmistakable evidences existed of the expenditure of energy in the ganglion cells. He thought the metaplast granules were the hitherto unrecognized evidence of this expenditure. In inveterate cases of epilepsy these granules would be found in the interior of the nerve cells and elsewhere.

Dr. Peterson said he had seen two or three cases of hemiplegia coming on during epileptic attacks, and they had been permanent.

Dr. Clark said that he had seen about forty cases of exhaustion paralysis in which the duration of the paralysis had varied from forty seconds to two or three weeks.

109. LÄHMUNG DES TRIGEMINUS UND ENTARTUNG SEINER WURZELN INFOLGE EINER NEUBILDUNG IN DER GEGEND DES GANGLION GASSERI; BEITRAG ZUR FRAGE NACH DER TROPHISCHEN BEDEUTUNG DES TRIGEMINUS (Paralysis of the Trigemini and Degeneration of its Roots in Consequence of a Neoplasm in the Region of the Gasserian Ganglion, etc.). Jarl Hagelstam (*Deutsche Zeitschrift für Nervenheilkunde*, Vol. 13, Nos. 3 and 4, p. 205).

Hagelstam compares his findings in a case of endothelioma of the base of the brain, causing complete destruction of the Gasserian ganglion, with those in a similar case reported by Homen in 1890. Hagelstam found important changes in the cells of the sensory and motor nuclei of the fifth nerve, and some alteration in the large cells of the cerebral root of this nerve. The sensory root and the spinal root were much degenerated. The retrograde degeneration of the cerebral root, and the alterations in the large cells near this root, were regarded as evidence of the motor function of these structures. The paralysis of the peripheral motor and sensory branches was complete, taste was lost on the anterior part of the tongue on the side corresponding to the new growth. The sense of smell on this side was also altered. The paresis of the soft palate, the impaired hearing, the changes in the pupil, and the paresis of the facial muscles, observed by Hagelstam, could not be attributed to the destruction of the Gasserian ganglion, as the tumor was quite large and involved more than the fifth nerve. The case shows that hemiatrophy of the face is not dependent on lesions of the fifth nerve, and that trophic fibers are not contained in this nerve.

SPILLER.

PHILADELPHIA NEUROLOGICAL SOCIETY.

March 27, 1899.

The President, Dr. James Hendrie Lloyd, in the chair.

Dr. Lloyd presented a young woman who had almost total internal and external ophthalmoplegia.

Dr. B. Sachs, of New York, remarked that instances of this complete form of ophthalmoplegia were, on the whole, rare. He had not seen a dozen cases in the course of many years. He thought that the diagnosis as made by Dr. Lloyd was entirely correct and that the case was of nuclear origin. Whether or not Hutchinson was right in believing that these cases were due to syphilis, is difficult to determine. He would not be so much inclined to ascribe them to syphilitic infection as he would some of the cases of partial ophthalmoplegia that are supposed to be nuclear.

A number of years ago Thomsen, of Berlin, described an unusual case in which there was specific infiltration of the oculo-motor roots, choking some fibers without destroying them, and giving rise to partial oculo-motor symptoms. That case had always stood out in his mind as an unusual example of root disease in contradistinction to what, on clinical grounds, would be supposed to be nuclear disease.

This case of Dr. Lloyd he was inclined to rank with those described by Wernicke under the head of polioencephalitis superior. In the majority of these cases, particularly in the acute form, there was a marked alcoholic history. Cases have been reported by the speaker and others, in which ocular symptoms were associated with poliomyelitis anterior, and it is evident that the affection may at times attack the gray matter in the floor of the third and fourth ventricles, or at other times involve the gray matter of the cord.

Dr. William G. Spiller said that although it was possible that the nuclei of the sixth nerves in the case were involved in the primary lesion, he would be more inclined to attribute the disturbance of function of the external recti muscles to impairment of the connection between the ocular centers by means of the posterior longitudinal bundles. The absence of any involvement of the facial nerves seemed to indicate that the sixth nuclei were not primarily diseased.

The case of Thomsen alluded to by Dr. Sachs resembled somewhat the one reported by Gibson and Turner (*Edinburgh Med. Journal*, 1897); in this a hemorrhagic infiltration was found in the oculo-motor nerve. A hemorrhage in the opposite frontal lobe was at first supposed to be the cause of the oculo-motor paralysis.

Dr. Spiller also referred to a case of hysterical ophthalmoplegia reported by Dr. Sachs.

Dr. James Hendrie Lloyd thought that the point made by Dr. Sachs in regard to root involvement was of interest. The possibility of this involvement could be excluded in the present case, because the third nerve was not exclusively involved. The fourth and sixth nerves were also paralyzed. In the absence of other lesions, the case could only be explained as one of those selective morbid processes involving the nuclei.

ERYTHROMELALGIA AND ALLIED DISORDERS.

Dr. B. Sachs, of New York, read this paper by invitation. While rare diseases are not necessarily interesting, the

reader referred to the importance of erythromelalgia, because of the discussion that necessarily involved the consideration of other disorders to which it is more or less closely allied. In his studies on the subject he attempted to answer the following questions: Does erythromelalgia constitute a disease *sui generis*, or is it a mere complex of symptoms which may be associated with a number of other diseases? Is it of peripheral or central origin? Is it always due to the same cause, or is it in any sense a purely nervous disease?

A brief review of the subject showed that the majority of writers who judge the matter from a purely clinical standpoint thought that the symptom-group of erythromelalgia was due to involvement of the posterior and lateral gray matter of the cord. This inference was based solely upon the association of erythromelalgia with such diseases as syringomyelia, Morvan's disease and tabes dorsalis. But one autopsy had been performed up to the present time on a patient suffering from erythromelalgia. In this case changes were found by Auerbach and Edinger in the posterior roots, but it is doubtful whether this disease of the posterior roots was at all responsible for the condition of erythromelalgia, while it may have had an important bearing upon the complicating tabes dorsalis. Examinations of sections of the peripheral nerves and of the arteries had been made by Mitchell and Spiller from an amputated toe in a typical case. Their findings seemed to bear out Mitchell's contention that while the disease may possibly be of central origin, it could also be explained on the assumption of a nerve-end neuritis.

In the patient detailed by Dr. Sachs the disease was typical for many months, until a large gangrenous ulcer developed on the dorsum of the foot. On this account amputation of the thigh, immediately above the knee, had to be performed, and opportunity was given for ample examination of the nerve strands and arteries in the leg. The histological examination which was made in conjunction with Dr. Alfred Wiener, of New York, proved that all the arteries of the leg, but particularly the popliteal and the anterior tibial, were markedly diseased. The lumen in the larger branches, as well as in the smaller, being almost entirely occluded. In the various nerves that were examined there was slight degeneration, altogether out of proportion, however, to the disease of the blood-vessels.

It is safe to assume that this obliterating endarteritis was present in the largest as well as the smallest blood-vessels of the thigh and leg. As the speaker's case was entirely uncomplicated, the findings prove that the symptom-group could be explained entirely on the assumption of peripheral and arterial disease, followed by nerve degeneration. He was thus

led to agree with Mitchell and Spiller as to the probable peripheral origin of erythromelalgia, laying greater stress, however, upon the disease of the blood-vessels than upon the disease of the peripheral nerves; and while it is barely possible that in some instances the same or similar group might be caused by disease of the gray matter of the cord, that proposition was still to be proved. Everything pointed, however, to the conclusion that erythromelalgia did not represent a *morbus sui generis*, but was a symptom-group which might exist independently of all others, but might at times be associated with other diseases.

The speaker finally called attention to the fact that there were conditions following upon chronic heart and arterial diseases which resembled erythromelalgia very closely, and that from the point of view of morbid anatomy, there was some resemblance between intermittent claudication and the disease under discussion, only in the former the obliterating endarteritis, as shown by Erb, leads to temporary motor paralysis, while in erythromelalgia the obliterating endarteritis causes a series of sensory and vaso-motor disturbances.

Dr. Wharton Sinkler remarked that the paper recalled to his mind certain cases that he had seen resembling erythromelalgia without pain. The symptoms were more those of arterial disease than of nerve disease. In some cases there was intense flushing of one foot when the limb was pendent. This led him to think that the theory of Dr. Sachs was certainly tenable; but the conclusion cannot be escaped that in typical cases of erythromelalgia, as described by Dr. Mitchell, there is primarily disorder of the peripheral nerves, as shown by pain, and that coincident with the pain, there are disorders of circulation which are due to the nervous disturbance.

The president asked if it might not be of value to institute bacteriological examinations in these cases. The extreme involvement of the intima of the arterial system indicated the possibility of some microbic infection. The fact that the condition was limited to the vessels of one limb would of course be an objection to this view. This would also militate against the idea of a central origin. The possibility of thrombosis in the main artery of supply should also be borne in mind.

Dr. W. G. Spiller thought that the presence of gangrene was not sufficient to exclude a diagnosis of erythromelalgia. If further examination shows that the arteries are usually affected in this disease we may expect to find gangrene occasionally. It seemed strange, however, that in the case reported by Dr. Sachs, as well as in the one reported by Dr. Mitchell and himself, the disease was asymmetrical. If arteritis were the only cause, or the essential cause, we would expect to find the limbs bilaterally involved in erythromelalgia. Dr. Sachs had stated that the arteries in his case were more diseased in portions remote from the area of gangrene; that would seem to indicate that the disease was hardly of microbic origin, so far as the gangrenous area was concerned.

Dr. Spiller said that the atrophy of the thigh muscles and the exaggeration of the knee-jerk, mentioned by Dr. Sachs, were interesting features of the case. Exaggerated knee-jerk was also observed in the case reported by Dr. Mitchell and himself. The atrophy of the thigh

muscles seemed to indicate that the anterior crural nerve was involved, and nevertheless the knee-jerk was exaggerated.

Dr. Spiller thought that Dr. Sachs had been very fortunate in saving his patient after so serious an operation, for surgical intervention in these cases has sometimes been fatal. In one case mentioned by Dr. Mitchell the patient died on the operating table, and in the patient reported by Dr. Mitchell and himself an ugly slough formed after the amputation of the toe.

The endarteritis described by Dr. Sachs agreed with the findings of Mitchell and Spiller, but Dr. Spiller was unwilling to assert from the findings of one case that the essential lesion of erythromelalgia was arteriosclerosis. This view may be correct, but it is exceedingly difficult to distinguish the primary lesion when nerves and vessels are diseased, as disease of either may lead to involvement of the other, and the tissues most involved are not necessarily the first affected. Dr. Mitchell and he had been unable to say which was the primary lesion in their case. Even if the process were primarily vascular, involvement of the nerves seemed to be necessary for the complete clinical appearance of erythromelalgia. In the recent thorough work on intermittent claudication by Erb, to which Dr. Sachs had referred, the symptoms of erythromelalgia were distinguished from those of intermittent claudication. Intense arteriosclerosis often exists without the signs of erythromelalgia.

Within the past six months Dr. Spiller had seen three cases presenting symptoms resembling those of erythromelalgia. One was a pedler who carried heavy weights on her arms, and walked all day. She complained of intense pain in her arms and hands, and said that her hands at times "got as red as blood." She suffered also from pain in her feet. The second case was that of a woman who stood nearly all day. She complained of much pain in her heels, and these parts were somewhat red. The third patient was a butcher, who complained of considerable pain and redness of the feet, and he had no knee-jerk, but no other sign of tabes was discovered.

The question had occurred to Dr. Spiller, Why do we not find symptoms of erythromelalgia in every case of neuritis? Possibly the disease is found only when the vaso-motor nerves are affected. It is not improbable that in different forms of neuritis different nerve fibers are diseased. We know that lead, alcohol and the toxin of diphtheria not only select certain nerves, but may select certain fibers within these nerves. Dejerine has shown that neuritis is not uncommon in tabes, and the ataxia may be in part due to this neuritis. It is not unreasonable to believe, therefore, that in some forms of neuritis the vaso-motor nerves are especially involved.

Dr. H. A. Hare said that he had not heard all of the paper, and did not know whether the gangrene was of the moist or dry variety, or whether the veins were affected as well as the arteries. From the occlusion of the arteries he should expect to find dry gangrene, whereas, if the veins were diseased, the gangrene would be of the moist variety. This had been the case in endarteritis in typhoid fever.

Dr. Charles S. Potts referred to a case that he had seen a number of years ago at the University Hospital. The patient, a young man, had one toe crushed by a stone. Shortly afterwards he developed ascending neuritis, and after a certain length of time this was followed by typical erythromelalgia.

Dr. A. A. Eshner thought that it was not a remote possibility that in erythromelalgia we had primarily to do with some toxic process, not necessarily a poison from without, but more likely a poison generated within the body. The objection arising from its

peculiar localization would have to be disposed of as Dr. Spiller had suggested. We do not know why it is that certain poisons have certain affinities. Many toxic substances expend a part of their activity upon the blood-vessels, and some also upon the nerves. In erythromelalgia there may be a degenerative process involving the blood-vessels and an inflammatory process involving the nerves, and through this vicious circle there results an aggravation of the symptoms attending either process. It seems not impossible that in this *combination* is to be found the explanation of the peculiar train of phenomena that we see in erythromelalgia—why with simple neuritis or with simple arteriosclerosis we do not have erythromelalgia, while with the association of neuritis *and* arteriosclerosis symptoms of erythromelalgia develop.

In addition to the cases referred to there was another that was under Dr. Mitchell's care, and in which operation produced a successful result. In this patient, a man of about twenty-five years of age, the symptoms followed a traumatism of the foot, induced by a falling stone. He was subjected to nerve stretching and nerve excision, and the symptoms at once improved, and ultimately disappeared.

Dr. Joseph Sailer referred to a case which he had recently observed and which was in the wards of Dr. J. Chalmers DaCosta at the St. Joseph Hospital. When the man stood the feet swelled and became of a bluish, dusky color. The pain was not excessive, but the swelling and pain were increased by walking. Pressure on the posterior tibial, the anterior tibial and the sciatic nerves caused intense pain. An interesting feature was a surgical complication which had occurred.

Dr. F. Savery Pearce called attention to the fact that sometimes cases of periostitis have been differentiated with difficulty from erythromelalgia. He referred to a case in which Dr. Mitchell was in doubt as to whether it was one of periostitis or erythromelalgia.

Dr. B. Sachs in concluding the discussion, said that no bacteriological examination had been made. The fact that the arteries were more diseased higher up than in the vicinity of the gangrene would be sufficient to disprove the microbic origin of the disease.

In regard to the slight difference between the view of Drs. Mitchell and Spiller and that expressed by himself, he wished to be understood in this particular; he did not at all deny the importance of neuritis as a possible factor in the disease. He sided entirely with Dr. Mitchell and Dr. Spiller as against the majority of German writers who have, without microscopical study, concluded that this was a central disease. He thought that symptoms of erythromelalgia may be associated with some central disease, but in the uncomplicated cases that have been reported it is not necessary to seek any central nervous disease—the peripheral changes are sufficient to account for the condition. He believed that erythromelalgia would be shown to be chiefly, or very largely, of arterial origin. The changes in the arteries are so much more pronounced than those in the nerves that he thought it was hardly possible to make the nerves responsible for these changes.

There was one point which he had neglected to mention. Several of those afflicted with erythromelalgia have been tailors, or those who sat in a crouched position or ran sewing-machines, and he thought that it might be better to go back to a simple mechanical explanation rather than to seek for a bacteriological origin. He was inclined to doubt the bacteriological or toxic causation of the disease which, after lasting for years, showed relatively slight changes.

In reply to Dr. Hare he said that the veins had been examined and found to show only very slight changes. In his case there was moist gangrene of the foot and also dry gangrene of the toe.

Periscope.

ANATOMY AND PHYSIOLOGY.

110. ON THE INNERVATION OF THE INTRACRANIAL VESSELS. A. Morrison (Edinburgh Med. Journal, 1898, IV, 5, p. 413).

Working upon the brain of the fetal cat, the author succeeded in demonstrating fine nerve fibers coursing with the vessels of the pia mater, twisting about them, and terminating upon them in plexiform manner.

In his paper he considers the subject under the following heads:

1. "The character of the structures from which the nerves in question rise."
2. "The size of the vessels in association with which the nerves have been found."
3. "The character of the nerve fibers."
4. "The mode of termination of the nerves in the vessels, and in the pia mater."
5. "The presence of ganglion cells in the stems of the innervating trunks, and the character of these cells."

Of the first, he has not been able to get a very good idea.

2. The size of the vessels in association with the nerves was from 10 to 20 micra. The nerves themselves bore direct relation to the size of the vessels, the larger being about 2 micra in diameter, the individual branches measuring about 0.5 micron.

3. The nerve fibers are interrupted at intervals by nuclear bodies. The nerves have sheaths showing connective tissue corpuscles in the larger branches, and hyaline in the smaller ones.

4. The nerves terminate, both in the pia mater, and on the vessels, in a plexus, whose ultimate ramifications end in points so fine that their exact nature cannot be made out.

5. The ganglion cells occurring on the nerve trunks are unipolar and from 1 to 2.5 micra in diameter. They have a well marked nucleus and nucleolus.

His specimens were prepared by Sihler's hematoxylin method. The article is illustrated.

ALLEN.

111. WEITERER BEITRAG ZUM VERHALTEN DER REFLEXE BEI HOHEN QUERSCHNITTSMYELITIDEN (A Further Contribution to the Behavior of the Reflexes in High Transverse Myelitis). Joseph Fraenkel (Deutsche Zeitschrift für Nervenheilkunde, Vol. 13, Nos. 3 and 4, p. 274).

Fraenkel reports a case of psammoma of the spinal cord, extending from the tenth thoracic to the first lumbar segment. The tumor seemed to arise from the posterior part of the cord. The twelfth segment was entirely destroyed and the first lumbar was not intact. The patellar reflex could not be obtained, but a tap on the patellar tendon caused reflex movement of the adductors.

About one hundred cases of complete, and about ten of partial, supralumbar lesion of the spinal cord have been reported, in which flaccid paraplegia existed, although the pyramidal tracts were degenerated and the lumbar cord and peripheral motor nerves were not diseased. Fraenkel believes, therefore, that the patellar reflex is lost in every case of complete supralumbar lesion, but he does not believe that

flaccid paraplegia of spinal origin is a proof of complete transverse lesion. Ten cases of lost reflexes from a partial lesion of the cord have been observed. Fraenkel gives the various theories which are held relating to the flaccid paraplegia from complete supralumbar transverse lesion, and accepts the views of Bastian, with some slight modification. He makes the preservation of muscular tonus of great importance in the maintenance of the reflexes. SPILLER.

112. LA NON-EQUIVALENCE DES DEUX HEMISPHERES CEREBRAUX (The Non-equivalence of the Cerebral Hemispheres). Klippel (*Journal de Méd.*, March 6, 1898).

After examination of a large number of specimens, the author finds a distinct difference between the secondary degeneration following lesions of the right and of the left cerebral hemispheres.

He has found descending degeneration of the crossed pyramidal tract after cortical softening on the left side limited to the frontal lobe anterior to the ascending frontal convolution, and leaving this entirely intact. In lesions involving the Rolandic convolutions he finds degeneration of the pyramidal tract more marked when the lesion is on the left side. Involvement of the direct pyramidal tract is more frequent in left-sided lesions. Bilateral descending degeneration, that is, degeneration of the crossed pyramidal tract on both sides from a unilateral lesion, is seen almost exclusively in lesions of the left side.

PATRICK.

113. AN EXPERIMENTAL STUDY OF VISIONS. Morton Prince (*Brain*, 21, 1898, p. 528).

This is an interesting paper on the genesis of visions with the related phenomena. The author has summed up the observations as follows:

(1) Visions in sane persons may be revivals of past visual experiences, which originally may have been conscious or sub-conscious. The original sub-conscious experience may have occurred in a moment of absentmindedness, or may not have been sufficiently intense to have entered consciousness, or (rarely), may have occurred in somnambulism.

(2) The vision, instead of being a revival, may be a newly created pictorial representation of past experience other than visual. That is to say, past impressions of one or more senses (touch, hearing) and actions may translate themselves into a representation by another sense as a vision.

(3) It is probable, though not proved, that a vision may not reproduce or represent any past experience, visual or other, but may be newly created out of something the subject has read, heard or thought. The inference from, and passing thoughts about, known facts, may weave themselves into visions. This was probably the origin of the visions of Joan of Arc, and religious enthusiasts.

(4) Visions may partake more or less of the characteristics of these classes, being partly revivals, partly representations of actual non-visual experiences, and also of the subject's knowledge, inferences and thoughts.

(5) Generalizing, it is possible that hallucinations of the other senses, especially of hearing, and as exhibited by trance mediums, may have a similar origin and composition.

(6) Analogous phenomena may be observed in the attacks of hysterics where the passing thoughts in the normal state may appear as insistent ideas in the attack.

(7) It is probable that thoughts which have strongly absorbed

the mind and expressed the longed-for ambition, or the ideas and beliefs of the subject, may appear as visions. The subjects then are apt to look upon them as inspirations. In this way have arisen the visions of political personages like Joan of Arc, Bismarck, and religious enthusiasts like Luther, Fra Angelico, Catherine of Sienna and others.

(8) Visions, artificially created, may be representations and revivals of the experience of the hypnotic personality, of which experience the waking consciousness has never had knowledge.

(9) Impressions on the sense organs which never entered consciousness (therefore neither known nor remembered) may afterwards appear as visions.

JELLIFFE.

PATHOLOGY AND CLINICAL NEUROLOGY.

114. PRIMARY DIFFUSE SARCOMATOSIS OF THE PIA MATER. Frey Svenson (Nordiskt Medicinskt Arkiv., 1897, No. 6; Progrès médical, April 9, 1898).

A patient of fifteen years died after having had symptoms of brain tumor for six years, when the autopsy revealed a diffuse sarcomatosis of the base of the brain, extending to the upper cervical region of the cord, but entirely confined to the soft membranes. There were also lobes of the growth found on the inferior surface of the cerebellum, in the right lateral ventricle, and in the temporo-occipital fissure. The growth, or growths, were supposed to originate in the endothelium of the subarachnoid space. The author was able to find in the literature fifteen cases of diffuse neoplasm limited to the leptomeninges of the central nervous system.

PATRICK.

- 115 PARASITISME DES CENTRES NERVEUX PAR MYCOSE (Mycotic Infection of the Nervous Centers) J. Roux & J. Paviot (La Presse Médicale, 23 Fevrier, 1898, p. 102).

The patient, forty-one years of age, was admitted to the hospital suffering from paresis of the lower extremities and other nervous disorders. She was married, had had one miscarriage and one child which died in early infancy. There was no neuropathic heredity, and, aside from excessive indulgence in alcohol, no history of previous disease. Fifteen days before admission to the hospital the patient caught cold during menstruation, and felt very much fatigued. The following day she had a slight attack of sore throat, and noticed abnormal sensation in the lower limbs, that is, tingling and stabbing pains. She was so weak that it was almost impossible for her to remain standing. Her condition continued to grow worse, and she applied for admission to the hospital. Her condition was as follows: Great weakness of the lower limbs; locomotion, however, was possible, but the patient walked with a staggering gait; the station was poor, the patient falling if not supported; there was no inco-ordination; the reflexes were lost; tactile sense was disturbed, the patient responding very slowly and localizing very poorly; the sphincters were intact. Two days later the patient was unable to stand, but could still move the limbs, while in bed. Sensation was almost completely abolished, particularly in the feet and legs, and the subjective pains and tingling were still present. There was no muscular atrophy, no fever, no disturbance of the heart or respiration, and the urine was normal. Three days later the paralysis had continued to ascend, and had involved the sphincters. The general condition, however, remained excellent. The following day

the arms were completely paralyzed, and one day later, on the twenty-second day of the disease, the patient developed tranquil delirium and some hallucinations. She complained particularly of a sense of anguish if she looked at any clear space of considerable size in her neighborhood, having a kind of phobia for empty space. Up to this time the heart had remained normal. The following day, bulbar symptoms appeared, and there was tachycardia, and on the twenty-fourth day the patient died. The most important changes in the central nervous system were: first, the marked increase in the consistency of the brain and medulla. The dura mater of the cord was covered by a multitude of little glistening tumors containing a clear substance. The surface of the cord was irregular and nodular, as if constricted by the pia mater, which formed a number of irregular rings about it, with projection of the substance between them. The microscopical examination gave some extraordinary results. In the pia mater, penetrating into the fissures of the cord, were found a number of micro-organisms of very unequal length, varying in size from that of the tubercle bacillus to long filaments following all the flexions of the pia mater. The latter were formed by chains of individuals placed end to end with a clear space between. Branches were not observed, nor were the ends swollen. These filaments were found in the lumbar region in the substance of the spinal cord, particularly surrounding the blood vessels. Similar, but fewer, bodies were found in the medulla and in the cerebellum. In the brain itself the results were doubtful; a number of small granules staining deeply by methylene blue, but not by Gram, were found around the blood vessels. In no part of the central nervous system was there any trace of inflammatory reaction or accumulation of round cells. Cultures and inoculations were unfortunately not made; but the authors are convinced that the parasite belongs to the group of streptothricæ. The symptoms presented by the patient resembled very closely those of Landry's ascending paralysis, and in the absence of inflammatory reaction, they assume that they were produced by the parasite in consequence of its mechanical inhibition of nervous action. The mode of entrance was probably by penetration of the mucous membrane during the attack of sore-throat.

SAILER.

116. DER MORBUS BASEDOWII (Basedow's Disease). Schwerdt (Münchener Medicin. Wochenschrift, 1898, Nos. 42, 43 and 44, p. 1334 *et seq.*).

As the author himself admits, the title of his article is somewhat misleading, since its aim is to show that Basedow's disease, myxedema and sclerodema all have their starting-point in the condition, enteroplosis. This last he defines as a constitutional disease—which could be more correctly called "general atony"—whose most prominent symptom is a ptosis of the abdominal viscera. The disease depends upon an irritable weakness of the nervous system, congenital or acquired, bearing a close relationship to neurasthenia.

The alteration in intra-abdominal pressure, through weakness of the muscles of the abdominal walls, and the atony of the stomach and intestinal musculature, tend to cause a stasis in the flow of blood and lymph, digestive disturbances, abnormal fermentations, and autointoxication. There is gradually more and more disturbance of the circulation in the abdominal lymphatics, the valves of the thoracic duct finally become insufficient, and the chyle tends to pass into the external lymphatics and to mix with the body lymph, hence the tendency to lymph accumulation at certain points, and the production of local-

ized edema, so common in Basedow's disease. By a further extension of this process, the author attempts to explain the production of exophthalmos, by lymph stasis in the orbital cavities.

On account of the circulatory disturbance, the heart becomes hypertrophied, and irritable, and beats more rapidly—the tachycardia. Finally the passages of toxins, and especially of the chyle through the thyroid gland—whose function, he thinks, is to separate from the blood, and to neutralize certain toxins—causes an irritation of the organ, hence a hypertrophy of it—the struma—and increased production of its thyreoantitoxin, so accounting for the other symptoms of Basedow's disease.

The process continuing, there are gradually produced induration and shrinking of the lymph glands, and changes in caliber of the larger lymph vessels, especially of the thoracic duct.

There is a tendency to dilatation of the lymph capillaries in certain regions, especially about the chest and upper extremity, and to accumulation there of mucoid material, causing local swellings. The thyroid gland atrophies, and ceases to functionate, and we have the clinical picture of myxedema. Sclerodema, which has many points of resemblance to myxedema, presents three stages, edema, induration, and atrophy.

Unlike in myxedema, the swelling does not spread to contiguous tissues, but occurs in independent foci. This, however, seems due to local causes, as injury, cold, rheumatism, etc. In both diseases, the author considers that there is a stasis of chyle and lymph, but while in myxedema this is mainly through obliteration of the thoracic duct, in sclerodema the obstruction is mainly in the lymph glands. The author quotes a number of authorities to support his opinions at certain points, but gives no post-mortem findings on which to base his theory, and relates no new facts.

ALLEN.

117. ON FOUR OBSCURE CASES OF INTRACRANIAL DISEASE. H. J. Campbell (Brit. Med. Jour., 1898, No. 1970, p. 959).

CASE I. A spinner, aged nineteen, had had headache for four months, vomiting for two weeks and dragging of the right leg for two days before admission to hospital. The next day paralysis of the right arm, aphasia and impairment of sensation on the whole right side were added. The patient was restless and irritable and the inner edge of either disk was found to be somewhat blurred; temperature 99.2, pulse 84, respirations slightly accelerated. For two days his condition remained much the same, when he suddenly died.

The necropsy revealed a most unusual cause for the symptoms. The left cerebral hemisphere was softer and more friable than the right, but no gross lesion was discovered except a few tubercles on the vessels at the base. Doctor Eurich (a very competent man) made the microscopical examination and found numerous tubercle bacilli in the gray matter of the motor area. The hemiplegia was therefore apparently due to pressure upon the cortical cells by the inflammatory edema, caused by the presence of the micro-organisms in this region.

CASE II. A woman of thirty-three, for a period of fourteen weeks presented the symptoms of tumor of the pituitary body. On examination *post mortem* no tumor was found, but an inflammatory thickening, principally a pachymeningitis, in the region of the sella turcica. As "here and there there were evidences of caseation" the lesion was probably of tubercular or syphilitic origin.

CASE III. A boy of fifteen complained of slight headache, a few minutes later vomited, after walking home vomited again, and in

doing so fell down unconscious. There was a slight general convulsion and he died six hours later.

At the necropsy, made thirty-three hours after death, the right half of the brain was found to be suffused with blood under the pia from the rupture of small vessels on the surface; the lateral ventricles, the third, and the third and fourth ventricles all distended with blood clot, the source of the hemorrhage being the choroid plexus apparently in the right ventricle, this being more distended than the left. The lungs were normal. The pericardium was universally adherent, the cavity being quite obliterated. The pericardium was also adherent to the adjacent lung and chest wall. The right ventricle was dilated and empty, the left being much hypertrophied. The spleen was large, but otherwise normal. The thymus was large. Both kidneys were very large, and were typical examples of congenital cystic degeneration. The stroma presented the naked-eye appearances of fatty degeneration. The bladder was large and thick-walled. The stomach was empty and somewhat dilated. All other organs were normal.

CASE IV. A woman, forty-two years old, subject to "attacks of acute hysteria" and with mitral stenosis, had "an emotional attack" while out driving. This was thought to be hysterical, but at the termination of the drive she was found to be semi-conscious, in which state she remained for some hours. On regaining consciousness she was found to have left hemiplegia, paralysis of both third nerves, was unable to talk and was very emotional. From this condition she gradually recovered, but had a second attack from which she died.

On making a necropsy the basilar artery was found plugged with an *ante-mortem* clot of quite recent date, and an old adherent clot on the anterior wall of the posterior cerebrals at their point of origin, occluding the vessels going to the posterior perforated space. There was also a recent hemorrhage on the surface of the right occipital lobe from the posterior cerebral. Except for mitral stenosis the other organs were healthy.

PATRICK.

118. NOTE SUR LES DÉLIRES D'AUTO-INTOXICATION ET D'INFECTION (Note upon the Delirium of Auto-intoxication and Infection). Regis (La Presse Médicale, Aug. 3, 1898).

Regis believes that the deliria of auto-intoxication resemble very closely those produced by other forms of intoxication, particularly alcoholism. He regards all these as being practically states of somnambulism, and cites an interesting case in which he was able to recall to the recollection of his patient his actions during the delirious state by hypnotizing him. This led him to employ suggestion for the cure of persistent post-febrile delirium, and he found that he was able to restore his patients in certain cases to memory, and in some cases to dissipate what had apparently become fixed ideas.

SAILER.

119. SECTIONSBEFUND BEI EINER FRISCHEN SPINALEN KINDERLÄHMUNG (Pathological Findings in a Case of Recent Spinal Infantile Paralysis). Matthes (Deutsche Zeitschrift für Nervenheilkunde, vol. 13, Nos. 3 and 4, p. 331).

The question as to whether anterior poliomyelitis is a disease which affects the ganglion cells of the anterior horns primarily or secondarily has never been positively decided. Charcot believed that the motor cells were primarily affected, and v. Kahlden has recently defended this opinion. All cases of anterior poliomyelitis, which have

been examined soon after the commencement of the disease, have shown myelitis. Recent cases alone are of value in determining the primary seat of the affection.

Matthes reports a case of anterior poliomyelitis in which death occurred eight days after the beginning of the paralysis. He found circumscribed hemorrhagic myelitis, with changes in the ganglion cells of the anterior horns, which he regarded as secondary. The vessels arising in the anterior spinal artery seemed to be almost the only ones affected. This is the first case of anterior poliomyelitis studied by the method of Nissl.

SPILLER.

120. UEBER EINEN FALL VON TUMOR CEREBRI (Concerning a Case of Cerebral Tumor). H. Oppenheim (*Deutsche med. Wochenschrift*, 10, 1898).

Oppenheim reports an interesting case of brain tumor. The patient spoke quite well so long as he was in the recumbent position, though he had some difficulty in remembering words. The disturbance of speech increased when he was sitting, and he had much difficulty in recalling words, and did not understand all that was said to him. Words which he could not remember when he was sitting were recalled almost immediately when he laid down. The disturbance of speech Oppenheim describes as amnesic aphasia and paraphasia, because the patient spoke much and sensibly, but had difficulty in finding words. In the sitting posture the disturbance of speech took the form of word deafness. A sarcoma was found in the left supramarginal gyrus at the necropsy, and involved also the posterior part of the first temporal gyrus. The left temporal lobe was supposed to have been pressed upon by the growth when the patient was sitting. SPILLER.

121. PATHOLOGY OF A CASE OF FRIEDREICH'S DISEASE. WITH A SUMMARY OF PREVIOUSLY REPORTED CASES. H. Mackay, M.D. (*Brain*, 21, 1898, p. 435).

The author presents an extended study of a case of Friedreich's ataxia and gives a very complete summary of all of the known published cases. He believes that our present knowledge of the pathogenesis of the disease cannot be better summed up than in Guizetti's conclusion, that Friedreich's disease depends upon a congenital predisposition, in consequence of which, in the early years of life, certain systems of fibers and nerve cells undergo a process of progressive atrophy, and that the process is independent of any contributory effect from vascular alterations. In further explanation of the process the author adds that the expression "certain systems of fibers and cells" in these conclusions of Guizetti ought perhaps to be understood as follows:

A. (Due to arrested development at the eighth month.) Tracts in the cord which are the latest to undergo medullation, viz., the pyramidal tracts, direct and crossed, and the postero-internal tract.

B. (Secondary to mal-development of Goll's column?) Systems of fibers and cells functionally related to the postero-internal tract. The extent of the atrophic process as to systems attacked and degree of degeneration in individual systems may be dependent upon the duration of the disease, these systems may include: 1. The peripheral sensory neuron complex in its entirety, viz., peripheral sensory fibers, ganglion cells, posterior roots and root zones (Burdach's, Lissauer's), fibers to Clarke's columns, to the middle zone, and to the anterior cornua.

2. Portions of the central sensory neurons, viz., cells in posterior horns, with associated ascending fibers, cells in Clarke's columns, with associated fibers (direct cerebellar tract), cells in middle zone, with

associated fibers (Gowers' tract), cells in medullary nuclei, clavate and cuneate, with associated fibers (internal arcuate). JELLIFFE.

122. PARALYSIE DU VOILE DU PALAIS ET DU FACIAL INFÉRIEUR DROIT, AVEC PARESIE DU PNEUMOGASTRIQUE ET DU PHRENIQUE DANS UN CAS D'ANGINE DIPHTHÉRIQUE (Paralysis of the Soft Palate and of the Right Inferior Facial, with Paresis of the Pneumogastric and the Phrenic Nerves in a Case of Diptheritic Angina. Varnali (La Presse Médicale, Aug. 13, 1898.)

The patient had suffered for some days from a sore throat, from which she apparently recovered, when she commenced to experience difficulty in gargling, and the voice became nasal. The soft palate was motionless and anesthetic, and failed to react even to electricity. A few days later there was a deviation to the left of the orbicularis oris and the right naso-labial fold disappeared. There was partial reaction of degeneration in the muscles of the paralyzed side. Three days later the patient suffered from tachycardia and difficulty in respiration, that was apparently caused by paresis of the diaphragm, which contracted poorly and irregularly. Deglutition was exceedingly difficult. The epiglottic reflex was lost, and the vocal cords were paretic. Subsequently there was tingling and a sensation of cold in the legs and arms, but she ultimately recovered. Varnali believes that the extensive distribution of the lesions in this case indicates the fact that the post-diphtheritic paralyses are due to neuritis and not to a central lesion. SAILER.

123. A REMARKABLE CASE OF APHASIA. ACUTE AND COMPLETE EMBOLIC SOFTENING OF THE LEFT MOTOR-VOCAL SPEECH CENTER (BROCA'S CONVOLUTION) IN A RIGHT-HANDED MAN; TRANSIENT MOTOR APHASIA, MARKED INABILITY TO NAME OBJECTS, AND ESPECIALLY PERSONS, CONSIDERABLE AGRAPHIA AND SLIGHT WORD-BLINDNESS. Byrom Bramwell (Brain, 21, 1898, p. 343).

An epitome of this interesting case is given in the title; the more complete history should be consulted in the original. The author's own conclusions are as follows: "It may, of course, be argued that the motor-vocal speech center is not necessarily limited to the posterior end of the lower (third) left frontal convolution (Broca's convolution), or indeed to the adjacent parts of the lower (third) left frontal convolution (for in this case the softening involved not only the 'foot,' but also the 'cap' and the 'orbital' portion of the third left frontal convolution), but that it may, in exceptional cases, of which this was an example, be situated in the adjacent parts of the left hemisphere (the lower end of the left ascending frontal convolution, etc.). In other words, that the modern view, that the lower end of the left ascending frontal convolution is merely the ordinary psycho-motor center for the lower face, tongue, larynx, etc., and that it is not, properly speaking, a part of the motor-vocal speech center, is erroneous. If this be granted, it must, of course, be allowed that it is unnecessary to suppose that the action of the left motor-vocal speech center was in this case taken up, and carried on by the corresponding center in the right hemisphere."

"In short, after the most careful and impartial consideration of the whole question, the most reasonable explanation of the absence of motor-vocal aphasia in this case, in which Broca's convolution was completely and acutely destroyed, seems to me to be that the function of the left motor-vocal speech center was taken up and carried on by the motor-vocal speech center in the right hemisphere of the brain."

JELLIFFE.

Book Reviews.

LEÇONS DE CLINIQUE THÉRAPEUTIQUE SUR LES MALADIES DU SYSTÈME NERVEUX. Par le docteur Gilles de la Tourette. Paris: E. Plon, Nourrit et Cie., 1898.

This volume of clinical lectures is in the French style, with which we all were so familiar during the life of Charcot. There is certainly no country except France, where it is so much the vogue to publish clinical lectures that a large volume of nearly 500 pages on rather commonplace themes would be looked upon as a matter of course. The volume exemplifies the evils of this kind of literature; it is diffuse without being exhaustive, and prolix without being especially learned. Withal it is interesting; and for those who take time to go through it page by page it will be found both instructive and refreshing to the memory. We cannot but protest, however, against a class of books which, like this one, require twice the time for reading that would be needed for a more condensed volume that could readily contain in one half the space all that this volume contains.

The subjects treated by Gilles de la Tourette are cerebral hemorrhage, neurasthenia, epilepsy, hysteria, tic douloureux, migraine, morphinomania, Ménière's disease, club-foot, syphilitic myelitis and locomotor ataxia.

On the subject of cerebral hemorrhage the author presents nothing new—and for this we certainly cannot criticise him.

Neurasthenia receives from Tourette a very satisfactory exposition. It is not only clearly outlined, but, what is of first importance, it is clearly differentiated from hysteria. This is what we would expect from an author whose monograph on hysteria is the best clear-cut description of this disease that we possess in any language. The author gives full credit to Beard for what he did to establish a recognition of neurasthenia, but he perceives plainly that Beard did not contribute much more than the name and had, himself, a very confused idea of the affection. With him it was a remarkable medley of hysteria, hypochondria and sexual perversion. Tourette subdivides the subject into cerebrasthenia and myelasthenia—not a bad observance, even if a little pedantic. He calls especial attention, even while distinguishing between the two neuroses, to the fact that hysteria and neurasthenia may coexist in the same individual, and to this combination he gives the name hysteroneurasthenia. This is characteristic of the analytical faculty, so well marked in the French mind, and which has made so many of their clinicians such able demonstrators of nervous diseases. It is certain that few American or English writers would proceed to such distinctions, and yet they are perfectly valid and even necessary. We miss, however, in Tourette's paper a clear recognition of certain neurasthenic states of mind which are described by Regis in his recent work as impulsive and aboulie obsessions. We know quite well that most authors include such mental stigmata under incipient or confirmed paranoia, but we are convinced from experience that Regis is correct in recognizing that such fixed or imperative conceptions may coexist with neurasthenia, *i. e.*, may help to constitute a true *cebrasthenia*. This is an important clinical fact which, it seems to us, is too much ignored by Tourette and others.

The lecture on hysteria is one of the most important in the volume, because it is devoted almost entirely to the therapeutics of this affection. No one is better qualified than Gilles de la Tourette to write

on hysteria, and his scheme of therapeutics, based upon an unrivaled hospital experience, is most interesting and instructive. His trenchant criticism of the theorists who claim that hysteria can readily be cured with hypnotism is especially noteworthy; hypnotism itself, he says, is nothing else than an hysterical crisis, provoked instead of being spontaneous. How easy it must be to confound the disease with the cure, and to overlook the vital fact that the patient who is readily cured by hypnosis is from that very fact proclaimed to be hysterical!

The only remaining subject in Tourette's book that we need note is syphilitic myelitis. The author devotes eighty pages to a discussion of this ever interesting if not ever fruitful theme. He is more optimistic about the curability of nervous syphilis than our own experience permits us to be—but this may merely be because he wants to put his best foot forward when he steps into print. His treatment, both of the theme and the disease, is conventional and open to but little criticism. The only fault, as already indicated, is that it is somewhat hackneyed, and hence disappointing. To read eighty pages from Paris, and to find that they are merely abreast of our best American thought and teaching, is flattering but not surprising, and is a little conducive to ennui.

JAMES HENDRIE LLOYD.

DIE NEUROLOGIE DES AUGES. Ein Handbuch für Nerven- und Augen-Aerzte. Von Dr. H. Wildbrand und Dr. A. Saenger. Erster Band, erste Abtheilung. Mit 63 Textabbildungen. Wiesbaden: J. F. Bergman, 1899.

An ophthalmologist and a neurologist have combined in what another reviewer aptly calls "the happy co-operation of two investigators, each an authority in his field," and produced a book that at once commands the earnest attention of workers in these two important branches of medicine, which have always been, and must continue to be, associated in their interests. Although this is the day of highly differentiated specialties, the union between ophthalmology and neurology is too intimate to permit the devotee of each to pursue separate pathways. One difficulty that this combined effort constantly encounters is the widely scattered literature, which makes orientation confusing. Hence, to gather the reports of cases and the records of autopsies bearing upon any given subject, and after collection to classify and analyze them, is a labor not only of love, but of a high scientific importance. This is the task which our authors have set for themselves and which, as far as they have gone, they have accomplished with skill and judgment.

The scope of their labors may be learned from the contents of the volume, which are as follows: (1) The position and form of the eyelids; (2) the form and width of the palpebral fissure under physiological and pathological conditions; (3) the lid reflexes and the anatomical condition of the orbicularis palpebrarum; (4) the associated movements of the lid and bulbus; (5) spasm of the levator palpebræ; (6) paralysis of the levator palpebræ superioris—ptosis. There is a full description of the anatomy, pathology and physiology of the eyelids, in so far as they pertain to the subject under discussion, and we find discussed such interesting topics as periodic edema and hemorrhages of the lids, spontaneous gangrene, irritation and paralysis of the sympathetic, the ocular phenomena of exophthalmic goiter, the associated movements of the orbicularis with the upward, downward and lateral rotations of the eyeball, as well as the associations between the movements of the lids and the pupillary reactions. In the last-named para-

graph the authors have failed to notice Gifford's observations on the orbicularis pupillary reaction, which observations, however, may have been published after the book went to press, as they are of comparatively recent date. There is an interesting paragraph on facial paralysis and palpebral phenomena in association with voluntary and involuntary innervation of various facial muscles. Cramp of the levator palpebræ is dismissed in four pages, which brings the reader to Chapter VI., with which the book concludes, in the fullest possible discussion of paralysis of the levator palpebræ superioris—that is, ptosis.

This chapter, a book in itself of nearly three hundred pages, is an amazing example of industry and bibliographical research, and its comprehensiveness may be imagined when it is realized that after a general consideration of the subject there are discussed congenital ptosis, cortical ptosis, isolated double-sided ptosis, nuclear ptosis following chronic disease, ptosis associated with asthenic bulbar paralysis, and finally, ptosis following nuclear paralysis which has resulted from subacute or acute morbid processes.

Nothing in the literature of this subject seems to have escaped the authors. In addition to the general text, a series of tables of condensed case histories is appended; for example, of ptosis associated with chronic progressive ophthalmoplegia which remained localized, ptosis occurring in tabes dorsalis without autopsy, ptosis in the same disease with autopsy, paralysis of the ocular muscles which occur with influenza, diphtheria, lead intoxication, botulismus, etc. This would seem to exhaust the subject of ptosis, but we are promised that the second volume of this book shall continue this topic in a discussion of this symptom when it is brought about by syphilis, cerebral hemorrhages, cerebral tumors, trauma, poliomyelitis, etc., etc.

The volume is more freely illustrated than is the custom of German monographs, sixty-three woodcuts being presented, for the most part reproductions of photographs of the various types of ptosis. We have nothing but praise for this book. It is a monument of industry which will always be consulted with profit, and which should be read by all those who are interested in the subjects of neurology and ophthalmology.

G. E. DE SCHWEINITZ.

MEDICAL JURISPRUDENCE OF INSANITY OR FORENSIC PSYCHIATRY. By S. I. Clevenger, M.D.: with an Exhaustive Presentation of the Judicial Decisions upon the Subject by F. T. Bowlby, 1898. Two Octavo Volumes. 1,300 pages.

One of the most frequent mistakes made in reviewing works of a scientific character is to devote the entire review to the shortcomings of the volume under consideration. The reader of reviews of a medical work has a very practical desire to ascertain if he is compelled to place the book among his acquisitions as a work of such importance that it is necessary to his library. It is, therefore, in justice to author, publisher, and purchaser that the volumes should receive their full modicum of praise and, if desirable, their measure of unfavorable criticism. Dr. Clevenger has given to the profession a valuable contribution, one which every one interested in the subject of medical jurisprudence should possess, and a work which every alienist should consult. It meets all the requirements of lawyers and physicians in civil and criminal cases. It is a complete bibliography and index-rerum of forensic psychiatry. It is a work of reference of great scope, and in its field the only one. As far as its special references to judicial decisions and points of legal adjudication generally are concerned, it is filled with thousands of cited cases which alone make it valuable.

In a work like this it is of little moment to differ with the author's conceptions of psychology or his point of view of degeneracy, habit-neuroses, and perversions; but it is of great practical importance to know that there is a work which covers so thoroughly and well the wide fields of forensic psychiatry. J.

GENERAL PHYSIOLOGY. An Outline of the Science of Life, by Max Verworn, Ph.D., Professor of Physiology. Jena. Translated from the Second German Edition by F. S. Lee, Ph.D., Adjunct Professor of Physiology in Columbia University, N. Y., London, and New York. Macmillan & Co. 1899.

It is not often in the busy scramble for the acquisition of facts that it is vouchsafed to one to be able to sit down and gather together the disjointed fragments of numerous observations and weave them into a fabric of general truths. Such a task, however, has been performed by the author of this present volume, and performed in a manner that has won the attention and the praise of the entire physiological world; but the work is not only for the physiologist, it is one for every thinking and active worker in the domain of the biological sciences. Verworn has given the physiology of the cell its full consideration, and has gathered together the truths respecting the elementary phenomena of life in a way that is singularly attractive and practical to those who would seek the broader and deeper interpretations of nature's phenomena. For the neurologist and alienist there is much of primary importance if he is to understand the elementary truths of the mechanism of life in both its physical and psychical aspects.

The work is divided into six chapters. These deal with: The Aims and Methods of Physiological Research, in which is considered the problem of physiology, the history of the research and the methods of Study: Living Substance, in which is treated its composition and the relations of living and lifeless substances: Elementary Vital Phenomena discussing the phenomena of metabolism, the causes and conditions of form changes, and the problems of the transformation of energy: The General Conditions of Life, taking up in turn the present conditions of life on the surface of the earth, its origin, and the history of death and decay: Stimuli and their Actions, which is a chapter of especial interest to the neurologist and alienist. The author here discusses the nature of stimulation, its varieties and intensity, the irritability of living substance, and the nature of life reactions. The problems of electrical stimulation, the influences of heat, sunlight, and other epiphenomena are severally and thoroughly presented: The Mechanism of Life is the title of the last chapter. It discusses the vital processes, the mechanics of cell life and the relationships of cell to cell and the interdependence of cell communities. An extensive bibliography completes the volume.

The translator has done his part in a very acceptable manner in that he has preserved the charm of the original, a feat that is by no means an easy task in a work of this kind. We predict for it a wide acceptance in its present form. The mechanical work of the publishers is excellent.

JELLIFFE.

AMERICAN NEUROLOGICAL ASSOCIATION.

Preliminary program of papers to be read at the twenty-fifth annual meeting of the association, to be held at the Hotel Dennis, Atlantic City, New Jersey, June 14, 15 and 16, 1899:

An address commemorative of the twenty-fifth anniversary of the association will be delivered by Dr. Wharton Sinkler, of Philadelphia.

Isolated Finger Paralysis. Dr. William Browning, of Brooklyn.

The Pathology of a Case of General Cutaneous and Sensory Anesthesia. Dr. Henry J. Berkley, of Baltimore.

Hemorrhagic Pachymeningitis of the Convexity, with Cervical Pachymeningitis Occurring in a Case of Tubercular Cerebral Tumor. Dr. Charles W. Burr and Dr. D. J. McCarthy, of Philadelphia.

Report of a Case of Asthenic Bulbar Paralysis. Dr. John Punton, of Kansas City.

Facial Paralysis of Unusual Distribution—Probably Congenital. Dr. F. W. Langdon, of Cincinnati.

Rigidity of the Spinal Column. Dr. Philip Zenner, of Cincinnati.

Two Cases of Muscular Dystrophy, with Necropsy (one of the Facio-scapulo-humeral Type, Reported Clinically by Duchenne of Boulogne, Landouzy and Dejerine). Dr. William G. Spiller, of Philadelphia.

Tumor of the Pituitary Body. Dr. George L. Walton, of Boston.

The Influence of Treatment in Paralysis of Ocular Muscles. Dr. William M. Leszynsky, of New York.

The Origin and Development of Obsessions. Dr. Smith Baker, of Utica.

Some Interesting Cases of Brain Disease. Dr. William C. Krauss, of Buffalo.

Tuberculous Myelitis. Dr. Joseph Collins, of New York.

A Case of Sub-dural Hemorrhage Causing Partial Word-deafness in which there was a Marked Inability to Name Objects and Persons. Site of the Lesion Disclosed by an Operation. Dr. Graeme M. Hammond, of New York.

Sensory Disturbances in Hysteria and Epilepsy. Dr. E. D. Fisher, of New York.

Anomia and Paranoia, with Report of Cases, and Remarks on the Sites of Lesions. Dr. Charles K. Mills, of Philadelphia.

Purulent Encephalitis and Cerebral Abscess in the Newborn Resulting from Infection Through the Umbilicus. Dr. Guy Hinsdale, of Philadelphia.

The Forms of Cerebral Meningitis. Dr. Charles L. Dana, of New York.

Hematomyelia and other Traumatic Affections of the Cervical Region of the Spinal Cord, including a Case Simulating Syringomyelia. Dr. James Hendrie Lloyd, of Philadelphia.

Notes on the Arrangement and Function of the Cell Groups in the Sacral Region of the Spinal Cord. Dr. B. Onuf, of Brooklyn.

A Case of Internal Hemorrhagic Pachymeningitis in a Child of Nine Years, with Changes in the Nerve Cells. Dr. William G. Spiller and Dr. D. J. McCarthy, of Philadelphia.

Some Considerations from a Clinical and Anatomical Analysis of one hundred and three Autopsies on Cases of Motor Aphasia. Dr. J. Frankel and Dr. B. Onuf, of New York.

Asthenic Bulbar Paralysis, with Report of a Case. Dr. Wharton Sinkler, of Philadelphia.

Tumor of the Medulla presenting Ataxia and Astereognosis as the Most Prominent Early Symptoms. Dr. F. X. Dercum, of Philadelphia.

The Unity of the Acute Psychoses. Dr. Philip Coombs Knapp, of Boston.

Landry's Paralysis. Dr. John Jenks Thomas and Dr. Philip Coombs Knapp, of Boston.

The Nervous Equivalents of Fever. Dr. Henry S. Upson, of Cleveland.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

MULTIPLE CAVERNOUS ANGIOMA, FIBROENDOTHELIOMA, OSTEOMA, AND HEMATOMYELIA OF THE
CENTRAL NERVOUS SYSTEM IN A CASE
OF SECONDARY EPILEPSY.

BY A. P. OHLMACHER, M.D.

DIRECTOR OF THE PATHOLOGICAL LABORATORY OF THE OHIO HOSPITAL
FOR EPILEPTICS, GALLIPOLIS, O.

An exceptionally rare and interesting combination of lesions characterizes this case, whose salient features are condensed and offered in this abstract:—

Synopsis.—Adult male. Secondary (Jacksonian?) epilepsy of comparatively recent origin. Spinal paraplegia, rapidly progressing. Terminal pneumonia.

Anatomical Diagnosis.—Right lobar pneumonia. Acute splenic tumor. Fibroendothelioma (psammoma) of cranial dura, pressing into Rolandic sulcus. Cavernous angioma of callosal gyrus, of optic thalamus and of cervical spinal cord. Hematomyelia. Osteoma of spinal arachnoid.

CLINICAL HISTORY.

From the notes furnished by Dr. Richard O'Connell, First Assistant Physician at the Ohio Hospital for Epileptics, who at-

tended the patient throughout the whole illness, the history is compiled as follows:—

Denis, S., laborer, widower, native of Ohio, was admitted to the Ohio Hospital for Epileptics, May 4, 1896, in his forty-eighth year. His father died in his thirty-fifth year of pulmonary tuberculosis; the mother of apoplexy in her sixtieth year. There were ten children in the family, of whom eight (six male and two female) are dead; three dying of tuberculosis, one of pleurisy, two of typhoid fever, one of diphtheria, and one of unknown cause. One brother is living and in good health. No mental or nervous disease is known to exist in family. Patient has one daughter living and well.

The patient has had diphtheria, "scrofulous" glands in groin, and erysipelas a year ago. His own statements as to the first appearance of the epileptic convulsions are contradictory. Once he stated that the primary attack appeared in his thirtieth year while working in a wheat field in very hot weather, when he fell unconscious and remained so for three days. Again, he said that the first fit occurred in his fortieth year, when they continued for three years, then abated for two years, to reappear in the fall of 1895 and continue till the present time. When he was in his twenty-fifth year he claims to have received a blow on the head, to which he sometimes charges the beginning of the epileptic attacks. From the patient's statement he has suffered both light and severe attacks; in the severe ones (which were only occasional) general convulsions occurred, the urine would be voided, the face become pale, and unless supported a fall would result. No information relative to a localized beginning of the spasms could be obtained, and it is not clear whether consciousness was lost in the severe seizures, for none of these attacks was ever witnessed by a physician either before or after admission to the hospital.

The notes of the entrance examination (May, 1896) give the height as 182.5 cm., weight 146 lbs., pulse 90, respiration 24, chest expansion 37-39 in. No evidence of pulmonary or cardiac disease is found. The bowels are habitually constipated, the appetite good. No mental disorder can be discovered; the patient being able to read and write, to converse intelligently, and his memory is fair. The patellar reflexes are prompt on both sides.

Up to the onset of the present illness nothing is recorded except an average of sixteen epileptic attacks during the first six months, then two attacks monthly for the second half year (while under mixed bromide and iodide treatment), followed by four months without a seizure, and two in the month before death.

About the first of May, 1897, the present illness began, when

it was noticed that the patient walked with a shuffling gait. He complained that difficulty was experienced in buttoning his clothes, and that there was a numbness along the ulnar side of both hands and forearms. The motion in these parts was still perfect, and rough tests, like pinching, pressing, and jerking the affected members, showed no absolute loss of sensation, while the grip was still perfect in both hands. On September 15 he went home for a visit, returning November 29, and stating that he had improved during his visit, walking six miles with little fatigue the day before making the return journey. It was now noticed that walking had become more difficult and that the patient required assistance in getting from the train to his cottage at the hospital, both feet and legs being dragged and moved with difficulty. November 30 a more careful examination showed that he could walk slowly only if supported, that the legs could be raised voluntarily only with great effort; but that the arms were still quite useful, enabling him to feed himself. There was no impairment of sensation, except on the ulnar side of both hands and forearms, where the anesthesia was quite pronounced. The patient says he has had aching in both legs for six months past, especially in damp weather, and also that spasms have occurred in the left leg. On the sixth of December a bloody diarrhea confined the man to his bed, when he was found to have well-marked motor paralysis in both legs, with anesthesia almost complete. Several days later a partial paralysis appeared in the left arm, followed in a day or two by partial paralysis in the right arm. From this date there was occasional involuntary jerking of both legs. The patellar reflexes are exaggerated, the anesthesia becomes more marked in the legs, gradually extends upward, and the bladder and rectum become involved, the urine dribbling constantly and bowels becoming obstinately constipated. Another examination was made December 19, when it was found that the motor paralysis of the arms had gradually increased, the anesthesia had gradually extended up to the trunk to the level of the second rib, while a total paralysis of the muscles below this point was present. On the left thigh, anteriorly, sensation was not absolutely lost, as pinching, pricking, pressure, heat and cold were all appreciated slowly. The tactile sense was abolished, except on the upper left thigh and radial side of both arms, where it was quite perfect. Pain at the level of the second rib radiating down the arms was complained of, and there was a line of hyperesthesia corresponding to the level of this rib. Deep pressure along the ulnar nerve gave no pain, but a sense of pressure. Pricking along the legs produced occasional spasms of underlying muscles, more marked in the right leg. The plantar, patellar, cremasteric and biceps reflexes in exaggeration could be

elicited on both sides. The flabbiness of the muscles of the arms and legs was noticeable, and some atrophy of them had occurred. The pupils responded promptly to light and accommodation, and the tongue was not deviated in protrusion.

A temperature of 100 degrees F. was now recorded, and the appetite was failing. December 20 the morning temperature was 100.6 degrees F., pulse 90; patient had slept but little; the urine was diminished in quantity; the same pains were complained of. In the evening the temperature was 101.2 degrees, pulse 100, respirations 28. An examination of the urine made on this date showed a specific gravity of 1.029, an alkaline reaction, but no evidence of sugar or albumin; eight days later another sample yielded a slightly acid reaction, a specific gravity of 1.021, and a trace of albumin which microscopic examination showed to be due to a moderate number of pus cells and spermatozoa. The next day (December 21) the morning temperature was 101.2 degrees, pulse 95, respiration 30; in the evening a slight fall was noted. Now the anesthesia was complete in the left thigh, the left arm had become almost useless, *but the reflexes were still preserved*. Beginning bedsores were found over the sacrum December 22, and the patient claimed he could feel the one on the left side, but not that on the right, which was more advanced. Both temperature and pulse had fallen slightly. On the 23d the temperature was 99.4 degrees and pulse 80; bedsores had appeared on the outer malleolus of the left ankle, and on this date a slight epileptic attack was noted, with spasms of the eye and mouth muscles and transient loss of consciousness. From the 24th to the 27th the conditions remained about stationary, but in the evening of the latter date a rise of temperature (103 degrees), of the pulse rate (115), and respirations (30) was noted. The next morning the temperature had increased to 105 degrees, pulse 120, and respiration 50; in the afternoon a fall of one degree in temperature occurred, with no change in pulse and breathing. The reflexes were becoming obtuse—the man was apathetic. He could still feel the passing of the hand along the radial side of the arms, and above the level of the second rib sensation seemed quite perfect. At four o'clock in the morning of December 28 he had a series of spasmodic jerkings of the arms and legs; lasting about an hour, with pain and excessive perspiration in the parts, and at this time he suddenly threw one of his arms across his chest, but could not return it, and jerked his legs above the level of the body, only to have them fall back helplessly. In the morning of December 29 the patient was found to be unconscious, temperature 105.8 degrees, pulse 120, respirations 36; the reflexes were all gone; the left side of the

chest was noticed to bulge. Death took place at noon on this date.

AUTOPSY.

At two o'clock in the afternoon on the day of death the immediate post-mortem examination was commenced, and the following data were obtained: The body is 183 cm. long, and in a fair state of nourishment. Rigor has not appeared. The skin is sallow, and is broken on the back by two large superficial bedsores, one on each side of the median line in the sacro-iliac region; a smaller superficial sore is also present over the left external malleolus; otherwise, no changes are noticeable in the skin, and a moderate pallor is alone noticeable in the mucous membranes. The left pupil is more dilated than the right. The left side of the chest bulges distinctly. A moderate fat-layer covers the trunk, and here the muscles are pale and flabby. About the lower lobe of the right lung a few adhesions of recent origin are found. The heart is arrested in diastole.

As to the thoracic and abdominal viscera it only is here necessary to record the existence of a croupous pneumonia of the right lower and middle lobes, in the transition of the red to gray hepatization, accompanied with the usual cloudy swelling of the other organs, and with a swollen and softened spleen.

The *skull* was of average thickness, and the skullcap was readily removed from the dura. As the hand was passed over the convex surface of the exposed cranial dura a hard mass was felt on the left side of the longitudinal sinus, in a position corresponding to the upper limb of the cerebral motor convolutions. The dura was cautiously stripped from the vertex so as to leave the region occupied by this hard mass undisturbed in its relations. No adhesions between the hard and soft meninges were found except in the region of the dural tumor, as the hard mass proved to be; here a few fibers served to bind the tumor to the pia beneath. The meningeal fluid was in no way changed, and no alterations could be detected about the base of the *brain*. The weight of the encephalon was found to be 1,385 grams. Without further dissection the whole organ, with its cap of dura, was placed in a large bulk of Zenker's fluid, where it remained for three days, to be then transferred to Müller's fluid, after having the cerebellum and stem cut away from the cerebral hemispheres, which were separated by a median longitudinal incision.

Except for the small tumor of the *spinal meninges* to be presently described, those membranes were unchanged. There is a slight increase of clear subarachnoid spinal fluid. Even when the spinal cord lies *in situ* a darkened area can be seen through the dura in the lower cervical region, corresponding to the level of the bodies of the sixth and seventh cervical and

first costal vertebræ. The cord is carefully removed from below upwards, retaining its dural sac about it, and only after its complete removal to a suitable flat surface is the dura incised. Now that the cord is exposed, its peculiar configuration in the lower cervical region is at once evident in the appearance of a well-marked fusiform swelling of this portion, together with the reddish-brown color of the thickened region, especially as seen from the dorsal aspect, which makes a marked contrast with the proper color of the spinal substance. For fear of disturbing important relations no incision is made into this enlarged portion, and the specimen is handled as little as possible, the only point elicited at this time being an

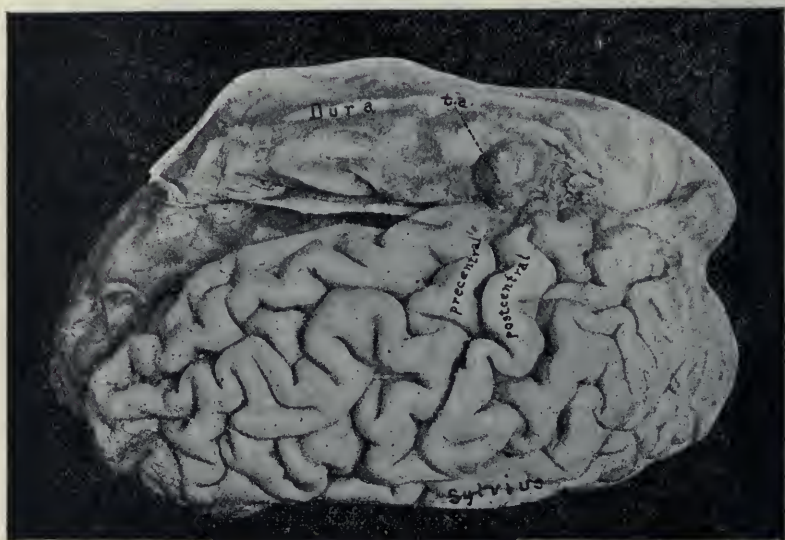


Fig. I. A photograph of the brain exposing especially the left cerebral hemisphere, from which the soft meninges have been stripped, and showing above, the dura turned back, with its tumor, *t.a.*, lifted out of its bed in the upper limb of the Rolandic fissure, where the depression marking its former presence still remains.

increased consistency of the swollen region as developed by delicate touch. The fresh cord is cut into sections 6-8 cm. long, and, with the dura remaining attached at the sides for support, is fixed for 48 hours in Zenker's fluid, washed, brought through ascending alcohols to that of 95 per cent., and left for future study. In several of the freshly-made sections below the region of the cervical swelling a tube-like excavation is noted, from which a thick, bloody fluid escapes.

Upon the completion of the hardening process various ad-

ditional examinations of the abnormal features in the central nervous system were made, which will be immediately discussed.

THE TUMOR IN THE CRANIAL DURA.

As soon as the preliminary fixation of the brain in Zenker's fluid was completed it was washed with water, the pia stripped from the left cerebral hemisphere, and before further dissection, photographed so as to show the dural tumor in its various relations. Figures I and II reproduce this photograph. The anterior end of the brain is to the left as one faces Fig. I. It will be seen that a portion of the anterior extremity of the right cerebral hemisphere, its convolutions covered with pia-arachnoid shows above the great longitudinal fissure. Resting on this right hemisphere is the dura, partially lifted out of the longitudinal fissure and turned back. At *t.a.* projects the tumor with which we are now concerned, appearing as a dis-

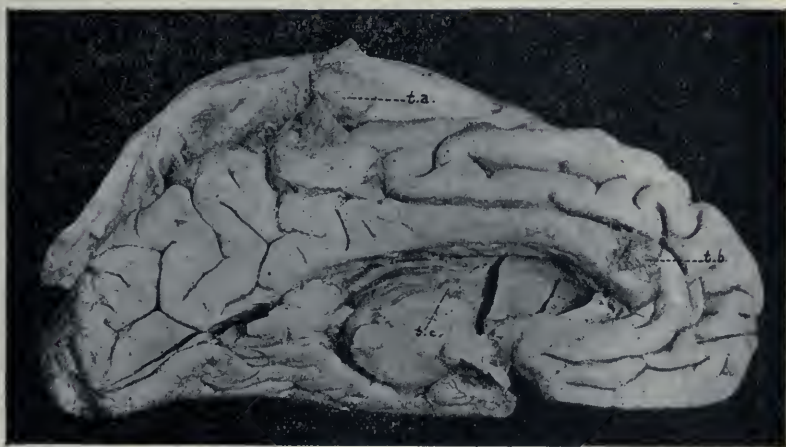


Fig. II. The left hemisphere, now seen from its mesial aspect, with the dura above it and turned back, and twisted in such a way as to bring the dural fibroendothelioma, *t.a.*, into view, held in place by a veil of pia-arachnoid hiding the paracentral end of the fissure of Rolando. At *t.b.* the external (mesial) projecting surface of the cavernoma in the callosal gyrus is displayed; while at *t.c.* is located the cavernoma in the optic thalamus.

tinctly rounded mass which has, in the turning backward of the dura to expose it for photographing, been lifted from its bed, a distinct impression of which still remains and is reproduced in the picture. This bed from which the dural nodule has been lifted is located in the superior end of the central (Rolandic) sul-

cus, which can be located in Fig. I. between the two central gyri. In Fig. II. another view of this dural tumor is seen at *t.a.*, this time from the mesial aspect of the left cerebral hemisphere, the dura being now drawn to the left and slightly twisted so as to bring the nodule into view, which, with the band of pia-arachnoid attaching it to the paracental lobule, hides the mesial end of the Rolandic sulcus.

These photographs record the location of the tumor and its general appearance better than mere words can, so that comparatively little need be said about the gross appearance of the neoplasm. As the figures show, it is a roughly spherical mass with a broad base of attachment to the dura on its inner or encephalic face, immediately to the left of the longitudinal dural sinus, at the side of the falx, projecting or growing freely downward; and so situated as to lie upon the upper end of the central sulcus, where a distinct bed or trough has been made by the tumor mass growing into the soft brain tissue, in which the end of the thumb may be snugly fitted. Owing to the perfect hardening the exact shape of this bed remains, even to several accessory depressions corresponding to small irregularities of the free tumor surface, so that the neoplasm fits into its bed as naturally as though it had been moulded in this position.

This depression on each side of the sulcus resulted, of course, from pressure-atrophy of the yielding brain tissue; and in this process the precentral gyrus, lying more directly beneath the hard tumor, suffered more, although the apex of the post-central gyrus also shared in providing a depression for the neoplasm. A comparison of these regions in the two hemispheres of the hardened brain brings out in a striking manner the extent and nature of this depression. On the right side the precentral gyrus reached the top of the brain as a well-rounded and prominent ridge, and the furrow marking its union with the superior frontal gyrus is sharply incised; but on the opposite side the rotundity of the upper extremity of the precentral gyrus is lost and the furrow obliterated to appear as a shallow bay. As previously remarked, several fine bands of adhesion extended from the pia-arachnoid to the tumor surface at the point where it depressed the brain substance, and these bands were severed in lifting the tumor from its bed. A glance at the figure (Fig. I) will show several spots in the top of the Rolandic sulcus at the location of the tumor bed, and this roughened region corresponds to the area over which the pia adhered to the proper brain substance so strongly as to leave erosions on being stripped.

The tumor proper measures 2 cm. in diameter, with its generally spherical contour broken upon the broad base of intimate attachment to the dura, where it is quite flat, due, no

doubt, to the resistance offered by the bony surface overlying this portion on the outside of the membrane. Towards the median line this base of attachment encroaches so closely upon the longitudinal sinus that it seems questionable whether an attempt at removal would succeed without tearing into this blood-space. Several accessory smaller, nodular projections serve to disturb the perfectly spherical outline of the free tumor surface, but there is no roughness or break in the smooth boundary surface except a few points where tags of adhering pia remain. Even in the fresh state the nodule was found to be quite dense, approaching the consistency of cartilage, but not becoming osseous. In the hardened specimen this density has not increased in proportion to the hardening of previously softer structures. An incision into the nodule shows it to be uniformly firm, and of the same faintly yellowish hue that is shown externally. As the knife enters the mass it grates perceptibly and a distinctly fibrous, or laminated, condition can be seen on close inspection of the cut surfaces. A slice 0.5 cm. in thickness was cut from the center of the nodule, and after proper preparation the paraffine-imbedded piece was sectioned and the sections stained.

The microscope morphology of this neoplasm shows its identity with the class of dural tumors called fibroendotheliomata. The ground substance is composed of rather coarse fibers running in various directions, and exhibiting the specific reaction of elastic tissue when stained after Weigert's recently published method; staining yellow with the writer's fuchsin picro-formalin combination; and red with picro-acid fuchsin. The coarser fibers of the stroma are homogeneous in appearance and contain but few much-elongated nuclei, while the finer fibers have more abundant nuclei, oval in shape and provided with a distinct chromatin framework. Lying in the ground substance are a moderate number of round ("sand bodies") masses, either homogeneous and glassy in appearance (calcareous), or distinctly laminated; in the smaller examples of which the cells composing the whorls can be readily seen in well-stained preparations. Along with the coarser elastic fibers and spherical bodies, the tumor contains a large number of spindle-shaped cells provided with quite large oval nuclei, and still larger cells with larger, more nearly spherical nuclei, in which the cell-body does not assume so decidedly a fusiform aspect. Small blood-vessels are present in the tumor parenchyma in moderate numbers, and their walls blend insensibly with the surrounding tumor elements. A reference to the micro-photograph reproduced in Fig. III will assist to make the description of the histology of this tumor clear.

Inasmuch as the entire encephalon had been fixed in the

same solutions, an excellent opportunity was afforded to note the effect produced by the pressure of the dural neoplasm upon the brain tissue beneath by comparing sections from similar regions in the two hemispheres. To this end a piece was cut from the upper limb of the left precentral gyrus in such a manner as to present on one side the surface indented by the pressure of the tumor, and on the other the mesial surface which had not been subjected to pressure. A piece of the same character was also cut from the top of the right precentral gyrus; then both pieces were carried side by side until sectioned. The treatment in bulk with Zenker's and Müller's

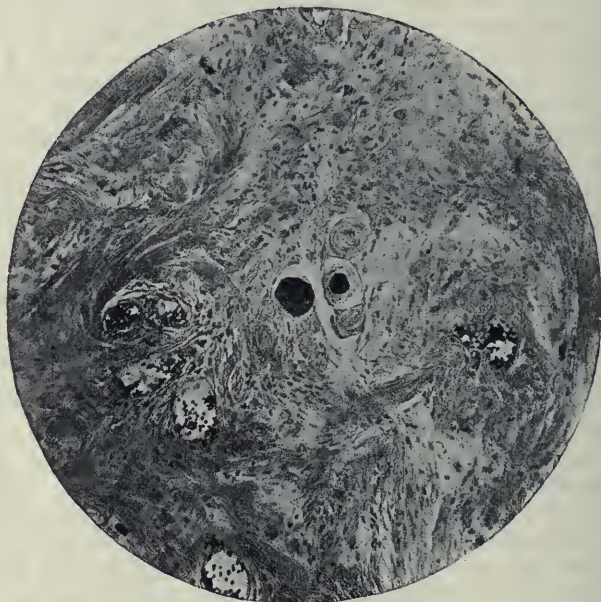


Fig. III. A microphotograph of a section of the dural tumor, *t.a.*, Fig. I, made with Leitz obj. 4, Zeiss compensation ocular 12. In the center are seen four "sand bodies," one quite homogeneous, the others showing more or less of concentric layers. The general fibrous and endothelial structure is reproduced, together with several blood-vessels containing red blood cells.

fluids was not well adapted for the preservation of the cytoplasmic structures revealed by Nissl's method; but, allowing for the imperfect results to be expected in this direction by the method followed, no marked difference in the cytological details of the cortical ganglion cells from opposite sides of the cerebrum could be found. With the *neuroglia*, however, the case was different, for upon the surface, exposed to pressure

and beneath it, a marked neuroglial reaction was to be seen in the increase of small, round or oval glia cells, and in a well-developed superficial gliosis. Even in a section from the left gyrus the contrast between the unexposed mesial (paracentral) and the upper exposed sides was most striking, for on the mesial, or paracentral, side the glia cells were little increased, while the superficial neuroglia was of ordinary depth, its component fibers staining poorly, and only in one or two places could hyalin spheres be found. Upon the depressed surface, however, the glia cells were more numerous, and here the superficial neuroglia was increased several times its ordinary thickness, while the network of glia threads composing the gliosis stained readily (with Unna's alkaline blue, for example); and this plainly demonstrable network of glia fibers held in its meshes numerous hyalin spherules. Directly beneath this gliosis the glia cells were increased in number throughout the thickness of the cortex, though the fibers did not stain prominently beneath the surface. Compared with the section of the right precentral gyrus, the characteristics of this gliosis in the area of pressure were still more sharply defined.

THE CEREBRAL ANGIOMATA.

After the division of the hardened cerebrum two more tumors appear in the left hemisphere, whose location can be seen by a reference to Fig. II, where they are designated as *t.b.* and *t.c.* While projecting above the surface, both of these new growths penetrate into and become a part of the brain substance, thus differing from the dural nodule. One of the tumors is considerably larger and more prominent than the other (*t.b.*); but in gross appearance they seem identical, being partly without the surface from which they spring and partly imbedded in it, rough and irregular on the exposed surface, and of a chocolate-brown color, contrasting strongly with the proper cerebral substance even in the bichromate-colored brain.

The first tumor (*t.b.*) is located in the anterior end of the limbic lobe. More specifically, it lies in the extreme anterior portion of the callosal gyrus, at the point where this convolution turns downward towards the base of the brain, its upper portion lying parallel with the anterior end of the calloso-marginal (prelimbic) sulcus, while its lower portion occupies the lowermost edge of the convolution, thus encroaching upon its whole width. The surface of this brownish protuberance is not smooth, for while a somewhat hemispherical outline is made by the projecting portion, this outline is broken by numerous small irregularities corresponding to accessory nodules, appearing quite black in the hardened specimen, which spring

from the larger mass. The stripping of the pia from the hardened hemisphere proceeded with perfect ease in the region roundabout this tumor, but stopped abruptly when its borders were reached. Here the soft meninges were bound so intimately as to be inseparable from the free tumor surface; indeed, they appeared to the eye to be what the microscope subsequently showed them to be, viz., *an integral part of the neoplasm*. At this point, also, the vessels in the pia-arachnoid were prominent, and several of them (branches of the anterior cerebral artery) ran over the tumor surface as small, tortuous tubes, one or more of which can still be seen by close inspection of the figure (Fig. II *t.b.*). The size of this external nodule is that of a rough hemisphere 1.5 cm. across, and it projects above the general level of the callosal gyrus about 0.5 cm. The external protuberance does not, however, mark the full extent of the tumor, for a series of transverse sections shows that it not only extends more deeply than would at first seem probable, but also reaches further antero-posteriorly than suggested by the projecting portion. It occupies fully both limbs and the knee of the anterior curved portion of the callosal gyrus, its inferior portion resting upon the transverse bands of white substance composing the genu of the corpus callosum, while its superior portion fuses with the pia extending into the calloso-marginal sulcus. Into the depth of the frontal lobe it passes a distance of nearly 2 cm. from the mesial surface, thus reaching through the medulla of the callosal gyrus and a little distance into the white core beyond. In consequence of its more extensive internal expansion this black tumor makes a roughly rectangular figure, with its long axis parallel with that of the hemisphere. The transverse cuts across the nodule, exposing the innermost portion, show at first glance black surfaces, but on closer inspection it can be readily seen that delicate bands traversed the cut surface, and in some places minute cysts are to be seen, doubtless corresponding to cavernous regions from which the black substance (blood-clot) had escaped during the section. In the cerebral substance bordering the larger tumor mass several accessory black foci can be seen.

Projecting abruptly from the prominent apical portion of the anterior tubercle of the left optic thalamus two or three millimeters posterior to the stria terminalis is the second tumor in the cerebrum to be seen at *t.c.* in Fig. II. Two knife cuts were made transversely across the left corpus striatum in the hardened specimen, the larger, posterior one at its lower end cutting across the stria terminalis; and behind the center of this cut the little tumor nodule can be located in the figure. It is somewhat similar to the tumor *t.b.*, but much smaller, measuring not more than 4 m.m. across. Like the larger

nodule, it lies half buried and half projecting from its seat in the thalamus. A much smoother projecting surface characterizes it, but its dark hue is like that of the larger callosal growth, making it readily seen. On being cut across it shows a series of black foci a millimeter or less in diameter, lying closely together, and separated by what looks like the yellowish substance of the thalamus. It is regretted that in making the section of this hardened brain no particular pains were taken in removing the choroid plexus from the left lateral ventricle, so that the relation of this little neoplasm to the vessels of the plexus cannot be determined. However, the tumor is situated on the thalamus at a point from which vessels return from this ganglion to reach the choroid plexus, and it is not difficult to assume a connection with the vascular channels of this region and the vascular tumor, as this growth proves to be.

For microscopic examination a slice about 0.5 cm. thick was cut from the neoplasm in the callosal gyrus by means of two deep frontal incisions, so as to include the full extent of the growth and some of the surrounding nervous substance. In the case of the smaller thalamic tumor it was completely removed from the hardened hemisphere, along with a surrounding border of brain tissue. Sections were cut from these pieces through different planes, and a considerable number from each tumor were examined after treatment with various staining combinations.

The callosal tumor proves to be a cavernous angioma (cavernoma), apparently having its origin in the pial vessels covering this portion of the cerebral hemisphere; at least, the surface of the growth is so intimate a part of the overlying meninx that no distinction can be made between them. That portion of the foreign mass which appeared black in the hardened specimen proves to be the angioma, and the little sacs noted in the macroscopic section are large blood-spaces from which most of the blood elements have escaped. The bulk of the new growth consists practically of three parts—blood-spaces, blood elements, and connective tissue. *A network of irregular blood-spaces* best describes the appearance of this, the principal part of the growth, and a striking variation in the size, together with extreme irregularity in shape, is also characteristic. The micro-photograph, Fig. IV, shows a number of the blood-spaces, of various sizes, as brought out with a low power. Within the irregular spaces are red blood cells, either diffused or aggregated in dense black masses. The region here shown is a point where the tumor encroaches upon the remains of the brain tissue. Some of the larger spaces attain such a size that the section of their wall quite fills the field of a No. 3 Leitz objective with ocular 3, while several of the smaller ones

may be found in a single field of the No. 7 objective with the same ocular. From a study of serial sections there can be no doubt as to the communication of the larger spaces—a matter of importance in differentiating this growth from a telangiect-



Fig. IV. Photograph with Bausch and Lomb obj. $2/3$ in., eyepiece 1 in., of a section of the cavernoma in the callosal gyrus (*ib.*, Fig. II) at the point where it borders the brain tissue, which can be seen below and on the right in the picture. The endothelial-clad blood spaces, both large and small, appear, filled with masses of blood elements more or less extensive and compact. The larger of these spaces by no means represents the extreme size attained by the largest caverns in these angiomas.

tasis. There is no regularity in the grouping of the larger and smaller spaces, but they are promiscuously mingled.

Both the larger and smaller cavities have a wall composed of endothelial cells, which appear as a homogeneous inner layer, with here and there a slight swelling corresponding to the situation of a fusiform nucleus. Sometimes this endothelial wall is the only structure bounding a cavity, and in places such thin-walled spaces lie side by side. More often the thickness of the endothelial lining is reinforced by a small amount of connective tissue composed of round or fusiform cells, and a delicate fibrillary network, or of coarser fibers of a homogeneous or hyalin aspect.

Even after the escape of many of these elements, as inev-

itably happened even in the most careful handling of the sections, the cavities in the tumor, both large and small, remain packed with red blood cells which have well preserved their shape. Scattered among these erythrocytes most of the spaces contain a moderate number of leucocytes. Several of the larger spaces show a central mass of polymorphonuclear leucocytes in the midst of red cells; and in a few of the medium sized cavities distinct thrombi, composed of polynuclear leucocytes and coarse intermingling fibrin threads, with but few red cells, fill the space. Such a thrombus is present in the larger space at the extreme lowermost margin in Fig. IV.

That a foreign mass of this size could grow in the cerebral substance and not produce marked effects upon the enviroing tissues is, of course, not to be expected, and it will, therefore, be no surprise to find that certain peculiar effects have been here produced.

Of course, a complete destruction of the nervous tissue at the immediate seat of the new growth has taken place, and in the nerve cells and fibers lying adjacent to the area occupied by the tumor distinct degenerative effects, like swelling and vacuolation; can be plainly made out. But the most marked reactive effect is to be found in the *neuroglia*, which has condensed itself at the periphery of the blood-spaces in the form of dense interweaving masses of fine glia fibers, interspersed with small round glia cells, and also containing *larger bodies looking like massive nuclei with an abundance of chromatin granules*. What the exact nature of these larger cells may be is not clear, but that they are in some way connected with the reaction of the neuroglia to the foreign mass is the opinion I hold. For the most part, the neuroglia fibers are the same delicate threads as seen in the superficial portion of the uninjured cortex in this region, but about some of the smaller and more isolated blood-spaces the fibers are considerably coarser.

In the brain tissue about the tumor the smaller blood-vessels are packed with red corpuscles, and in certain regions a number of more or less spherical hyalin bodies are scattered. Still another class of cells could be seen, both in the fibrillary connective tissue between the blood-spaces, and in the condensed connective tissue from the pia which in places separates the cavernoma from the mother tissue. These are rather larger, mostly spindle-shaped cells, whose substance is filled with coarse, yellowish or brownish pigment masses. From the general appearance of these cells, and especially after a study of them in other situations in this case, it is concluded that they are phagocytes, and that the pigment is hematic in origin, resulting from the destruction of the immense numbers of red blood cells lying in and about the cavernoma. The final verifi-

cation of this notion by the application of microchemical tests for ferruginous blood pigment was prevented by the preliminary chemical treatment of the tissue in the fixing and hardening processes.

Turning now to the tumor in the optic thalamus, it is found to be very similar to the one just considered in its microscopic anatomy, for it is made up of a series of blood-spaces with endothelial walls, separated either by a small amount of hyalin connective tissue, or by condensed neuroglia. The main difference in the structure of this smaller angioma is that it contains fewer cavities, and that these cavities do not lie so closely in contact. The black foci noted in the gross specimen proved to be cavities lined with a layer of endothelium and filled with blood elements. Of these larger spaces about ten can be counted in the sections, and one of these, the most superficial in situation, is of such a size as to nearly fill the field of the No. 3 Leitz objective, with the 3 eye-piece; the others being somewhat smaller. Then there are a number of smaller cavities not to be seen without the microscope, about fifteen or twenty in number, scattered between the larger ones. Distinct evidence of the fusion of several smaller blood-spaces to form it can be seen in the superficial large cavity when it is traced through several serial sections; but in the smaller ones the indications of fusion are not so clear. The blood elements in these cavities are mostly composed of red cells, with a few scattered leucocytes; an accumulation of leucocytes and fibrin threads (thrombus) being found only once upon the wall of one of the larger spaces.

With the spaces of the neoplasm less numerous and more perfectly separated, it is natural to suppose that the intervening neuroglia would be well developed—this is exactly the condition, for the supporting tissue of this portion of the brain surrounds the angiomatous cavities as though to insulate them from the remainder of the brain. In the description of the gross specimen it was noted that the surface of the tumor was smooth. This is found to be due to the fact that a layer of neuroglia invests the projecting portion of the neoplasm, and that the ependymal cells are present in part, but not to be found over the highest portion of the growth, where the enveloping layer is thinnest. Possibly the ependymal cells were lost in handling the specimen, but a more probable view is that they have been lost through the pressure. Large oval bodies containing a granular material staining with nuclear dyes like chromatin are found in the neuroglia in certain regions about the blood-cavities, quite like those seen about the tumor in the callosal gyrus.

THE NEOPLASM IN THE SPINAL CORD.

As soon as the fixing and hardening process was completed the examination of the spinal cord was renewed, at first with the undivided portions of the cord, and afterwards by coarse serial transverse sections. The cervical enlargement noticed in the fresh specimen remains, with its fusiform shape well pronounced and its point of greatest thickness between the origin of the seventh and eighth cervical nerves, from here tapering in both directions. This spindle-shaped enlargement is 3.5 to 4 cm. long, occupying quite exactly that portion of the cervical cord which is naturally largest. The oval shape of the normal cord is retained, and the measurements of a transverse section of the thickest portion of the spindle gives 2.1 cm. transversely (right to left) and 1.6 cm. antero-posteriorly (dorso-ventrally). Along the ventral aspect of the enlarged portion its extent cannot be

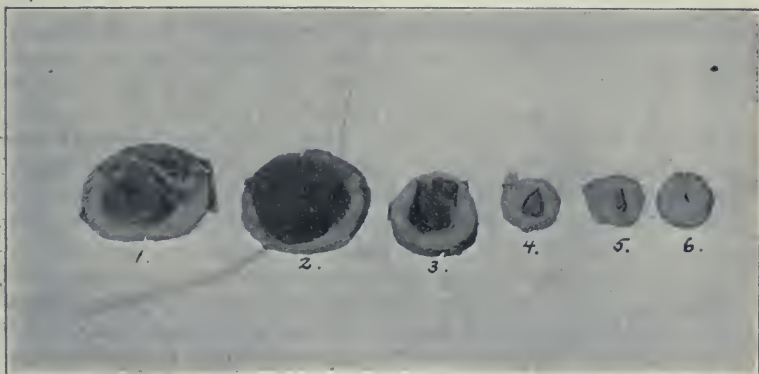


Fig. V. A series of macroscopic slices of the lower cervical, and thoracic spinal cord, showing at 1 the cavernoma in the upper right hand segment, and in the other figures the spinal blood-tube (hematomyelia) as it appears in various levels of its longitudinal extent.

so easily appreciated, but on the dorsal surface the point of departure of the cephalic end of the swelling is clearly marked, beginning abruptly in the space between the dorsal roots of the fifth and sixth cervical nerves. Towards its caudal extremity the swollen region tapers so gradually as to blend insensibly with the thoracic cord. This swelling is all the abnormality that can be noticed in examining the exterior of the hardened cord, for the opaque, greenish color of the specimen has entirely masked the dark hue which was apparent in the spindle-shaped mass when fresh.

When transverse macroscopic sections of the hardened cord are made, very instructive pictures are obtained. These are in part reproduced in the series of photographs composing Fig. V, where several sections of various levels of the cord are shown in nearly the original size in the photographs 1 to 6. The illustrations 1, 2 and 3 represent sections of the swollen cervical portion, 1 being the highest and corresponding to the level of the sixth cervical segment; 2, a section about 1 cm. caudad to 1; while 3 cuts across a level about 1.5 cm. below, and marks the point where the cervical and thoracic portions blend. A marked difference in the appearance in the uppermost sections, as compared with those lower down, is retained even in the photographs, as will be seen in comparing 1 and 2. Beginning at the point where the eye shows the swelling to abruptly commence in the cervical region (the level between the fifth and sixth cervical segments), a mottled appearance of the section is evident, produced by the presence of a number of dark brown or black foci, separated by thin tracts of lighter appearing tissue, recalling perfectly the appearance shown by macroscopic sections of the cerebral hemangiomas just described, and to be seen in the upper right hand portion of 1, Fig. V. Judging from its naked-eye appearance, this portion of the foreign mass in the cord is an angiomatous new growth, and this it proves unquestionably to be when studied histologically. The whole extent of this vascular tumor is much less than that of the enlarged cervical spindle. It begins abruptly at the cephalic extremity of the fusiform swelling, intimately in contact with the pia of the dorsal portion of the cord, and somewhat to the right of the middle line. At this point the tumor proper attains its maximum thickness, about 1 cm., and is the only occupant of the enlarged cord; but a few millimeters caudad to this point the angioma contracts more closely to the dorsal and right-hand side of the cord, while a uniformly dark mass makes up the larger part of the foreign material. A bit of the substance composing this black or brown mass is removed, and found to crush easily, and to be composed exclusively of blood elements, in which excessive numbers of well-formed red corpuscles predominate. So that this uniformly black mass, which, in sections more posterior, makes up exclusively the foreign material in the swollen cervical region, is really a great blood clot, and as such it will hereafter be designated.

Ignoring for a moment the blood clot which so materially aids in producing the enlarged condition of the cervical cord, it is found that the tumor proper extends but about 1 cm. in the long axis of the spinal cord, making this new growth an imperfect ovoid 1 cm. long and the same across, lying in the

dorsal¹ portion of the cord, somewhat to the right of the middle line, and intimately associated with the pia covering the dorsal convexity of the organ. With this subtraction, the remainder of the foreign mass making the cervical swelling is found to be nothing more than the massive blood clot, making the lower half of the deeply shaded portion shown at 1 and constituting the entire dark mass seen in sectional view in 2. At 3 which corresponds to the level of the first and second thoracic segments, the blood clot is broken into several portions, to become solid a little lower down, where it fills the tube soon to be described.

As seen in the case of the cerebral tumors whose gross appearance is identical with the neoplasm in the cord, the contrast between the dark brown or black tumor mass and the greenish nervous substance of the hardened cord is very sharp, so that no trouble is found to mark its location, which in the region of the cervical swelling corresponds, of course, to the portion unoccupied by the neoplasm and by the blood clot. This partial rim of condensed spinal marrow, thickest in the ventral aspect, is readily seen even in the photographs composing 1 and 2, Figs. V, because of its lighter shade; and the scantiness of this rim, and the almost complete transverse obliteration of the cord by the foreign mass can now be understood.

With due allowance for the increased amount of connective tissue arising from the pia and the pial septa of the spinal cord, the microscopic anatomy of the tumor proper is practically the same as that of the cerebral cavernous angiomas, thus confirming the anatomical diagnosis which made the three neoplasms identical. The actual tumor in the enlarged cervical region is a cavernous angioma, and, from its fusion with the pia over a considerable area corresponding to the right dorsal region, which the microscopic examination sustains, there is little reason to doubt that it owes its origin primarily to the blood vascular channels of the spinal pia, either from the layer originally enveloping this locality, or from the septa. The description given of the cerebral hemangiomas as a series of en-

¹ *Opposite to ventral*, not to be confused with "dorsal," as often used for "thoracic" spinal cord.

To appreciate more accurately the lesions in the cord which the sections in Fig. V depict, it should be noted that in 1 the section is viewed *from behind*, which brings the right and left sides of the picture to coincide with that of the specimen. But in 2, 3, 4, 5, and 6, we are looking at the sections from *in front*, so that the lateral halves are the reverse of those in the diseased cord. In all cases, however, the sections were so arranged in photographing that the ventral (anterior) face of the cord is represented in the lower border of the figures.

endothelial-clad blood-spaces of varying size, communicating in certain places to form extensive cavities, accurately fits the histology of the spinal tumor. In some places the thin-walled cavities lie closely in contact; in others they are separated by considerable connective tissue whose cells show signs of activity, especially that of a phagocytic nature in engulfing red blood corpuscles and disposing of masses of blood pigment; in still other regions of the tumor the spaces are more widely separated, the intervening tissue being either of connective tissue origin, or composed of the remains of compressed nervous tissue, or of neuroglia. Most of the spaces are packed with red blood cells, with here and there threads of fibrin, leucocytes, and large protoplasmic bodies filled with what looks like red corpuscles in various stages of dissolution (phagocytes). A few of the moderate-sized spaces are packed with masses of leucocytes, in which fibrin threads are mingled. Roundabout the neoplasm the blood-vessels, both of the external pia and of the remaining cord substance, are choked with erythrocytes. The process of phagocytosis, of which mention has already been made in connection with the angiomas of the brain, is most active in the cord, not only about the tumor, but throughout the hemorrhagic region above and below the tumor (the hematomyelia). Such an extraordinary amount of extravasated blood as was found in the spinal cord made, of course, a foreign mass of large proportions composed of red blood cells in various stages of disintegration and their pigmented substances, fibrin, and cellular detritus of the damaged cord tissue—to dispose of the foreign material was the first task of the reparative process, and there was abundant evidence that such work had made considerable progress at the time of death. As to the source of all the cells engaged in the work of removing the foreign substances, it is impossible to decide, but that the cells in the perivascular adventitial lymph spaces are actively participating there can be no doubt, for about each of the smaller vessels near the tumor, and alongside the blood tube, a sheath of new endothelioid cells is to be seen in the adventitial lymph spaces, and the larger of these cells have their protoplasm loaded with masses of blood pigment or with more finely granular foreign material.

Still directing attention to the cervical enlargement, it remains to consider the above described dark brown or black mass, making the bulk of the swelling, and which was suspected of being hemorrhagic in nature. On examining sections such was found to be the case; the whole mass, which fully occupied this region, except a narrow remaining rim of spinal substance, being one immense blood clot in which red cells largely pre-

dominate. The immediate layer separating this clot from the rim of cord substance is composed in part of connective tissue well supplied with small vessels, and in part of condensed neuroglia, the connective tissue probably arising from the remains of that which once occupied the fissures and septa of the cord; while the glia is that of the cord, which may have proliferated to some extent to meet the emergency caused by the presence of the foreign body. But of proliferation of neuroglia there is no pronounced evidence, as the glia fibers are not thicker than those ordinarily found in the cord, and they are not so abundant as one would expect to find in a sclerotic process. Surrounding the vessels in the wall bounding the blood mass are numerous large and active phagocytes, and similar cells are abundant in the tissue spaces. Outside of this enveloping layer the shell of damaged cord substance is found, with little to indicate its neural character except the presence of glia fibers staining nicely with gentian violet (gentian violet, picro-acid fuchsin), and here and there a distorted remnant of a ganglion cell, and nerve fibers, mostly swollen and distorted. There is no evidence, in examining sections from various localities in the cervical spindle, that any number of conducting fibers escaped destruction; in fact, it is altogether probable that from their large size and the extensive territory occupied by them, the angioma especially with the aid of the blood clot, *effected a complete transverse section of the cord in the cervical region*. No evidences of the central canal are to be found in the enlarged portion of the cervical cord.

THE BLOOD TUBE IN THE SPINAL CORD.

Both above and below the swollen cervical region, whose description has just been finished, a tubular excavation of the cord substance is found. Cephalad to the angioma the excavation extends a distance 4 to 4.5 cm., following approximately the left dorsal horn, and appearing first as a triangular slit, then roughly cylindrical and gradually becoming smaller and slit-like until only a little dot marks its location close to the point where the cord expands into the medulla. A chocolate-brown substance fills this excavation except where it has fallen out in handling the hardened cord, and this substance proves to be a mass of blood, mostly composed of red cells. Below (caudad to) the enlargement in the cervical cord the blood tube extends longitudinally a distance of 17 to 18 cm., and some idea of its appearance may be gained by referring to 4, 5, and 6 of Fig. V. The section from which these illustrations were prepared were all made in the thoracic cord, 4, being about 5 cm. below the cervical spindle; 5, 5 cm. further down, and 6, still 5 cm. further. Remembering that the sections lie ventral side downwards, and that they are viewed

from behind, these figures will convey a clear idea of the shape, character and extent of the hemorrhagic excavation in the thoracic spinal cord. A tendency of the false canal to seek the middle line can be seen, particularly in the more enlarged extremity, but throughout its extent it favors the left side, its tendency being to follow crudely the left dorsal horn of the thoracic cord.

The section shown at 3 represents the transition of the massive clot in the cervical swelling to the smaller tube of the thoracic region, and its peculiar appearance is due to the presence of several clots (appearing lighter in hue) in the dark mass of blood. The walls bounding the canal are at first roughly triangular in outline; then they are compressed laterally, making more of a slit as the excavation is followed down. As seen in the case of the tube above the cervical swelling, that found below it is also filled with a chocolate-colored mass composed mostly of red blood corpuscles.

It is necessary to briefly note several points in the histological examination of this spinal blood tube in sections taken from different levels. In all of the sections below that pictured at 3, which is really the tapering portion of the massive clot in the cervical spindle, the central canal could be found, its ependymal cells intact. This is also true for the canal extending cephalad from the angiomatous region, thus showing conclusively that the excavation did not choose the central canal for its extension. Another matter of importance is that there is *little neuroglial proliferation* about the lumen of the false canal; and only in places can a trace of connective tissue be found, and this does not encircle the tube. Even the condensed condition of the neuroglia which was seen especially in the case of the cavernoma in the optic thalamus, is here absent. There is a boundary made up of the neuroglia, but its fibers are not much in excess of those found in the unaltered regions of the cord.

In this absence of a well marked gliosis this excavation differs decidedly from an ordinary syringomyelia. Indeed, the appearance of this blood-filled tube was such as to suggest a recently produced mechanical excavation in which the pressure of escaping blood was the prime factor, the whole process being of so recent an origin that the ordinary attempts at repair, like condensation and proliferation of the neuroglial tissue, had not time to occur.

At one or more points in the periphery of the spinal blood tube groups of small blood-vessels, filled with corpuscles, were found; and about these blood-vessels, either in their advential and perivascular lymph spaces or the tissue immediately round about, large, oval, phagocytic cells, loaded with pigment, were seen.

Filling this false tube throughout its entire length is a mass

of blood clot composed of red corpuscles in all stages of disintegration, blood pigment, leucocytes, and fibrin in small amount. Large cells in which the outline of red blood cells still could be seen were also present in the blood mass in moderate numbers. The usual pressure effects could be detected in the tissues lying close about the blood tube, both in the ganglion cells and in their nerve fibers.

THE TRACT DEGENERATIONS IN THE SPINAL CORD.

With a tumor in the substance of the cervical spinal cord involving a portion of its substance, and a massive hemorrhage, making an almost complete severance in this region, the usual ascending and descending degeneration of a section of the cervical cord would be expected. Even in the macroscopic slices of the hardened cord the pallor of the pyramidal tracts below the tumor and of the dorso-mesial tracts above it show that such degenerative changes have occurred. Microscopic sections, especially those stained by the gentian violet, picro-acid fuchsin method, demonstrate these abnormal conditions in the columns more clearly. Above the cervical enlargement the evidence of degeneration in the dorso-mesial (Goll's) column is strongly marked, while that in the direct cerebellar tract is less evident, though partially developed on the right side. Below the cervical region, especially in the lower dorsal portion (where the blood tube does not interfere), the degeneration of ventrolateral (pyramidal) tracts is plainly to be seen.

THE OSTEOMA IN THE SPINAL ARACHNOID.

Had it not been for the preservation of the spinal membranes, by allowing the incised dura to remain as a side support for the hardening cord, the specimen now under consideration would probably have been overlooked on account of its small size. It was discovered after the dura had been stripped from the hardened cord, situated beside the point of exit of one of the lower thoracic nerves, where it appeared on the internal face of the dura as a white, spindle-shaped mass about 1 centimeter in length, and 3 to 4 millimeters in diameter. A striking resemblance to a bundle of small worms is shown by this little tumor when closely examined, and with a simple magnifier it is seen to be made up of a series of convoluted, rod-like bodies. It is closely applied to the nerve bundle, but not a part of it, for it can be separated by gentle lifting, when it is found to be connected with the sheath of arachnoid which surrounds the escaping nerve prior to its penetrating the dural membrane.

Running through the center of the convoluted mass, as viewed with the dissecting microscope, is a stem which passes parallel to the nerve bundle and *anchors to the dura*. More

Careful examination of the magnified object shows that the vermiform appearance is caused by the projection of small rod-like bodies from the central stem, out of which they grow in all directions, to end in knobbed extremities and show irregular thickenings, giving them a gnarled look. Both the stem springing from the dura and the worm-like tumor mass were very firm, giving the impression either of osseous, or of extremely dense fibrous, tissue. However, on account of its small size, moderately thin sections of the imbedded tumor mass were obtained, a circumstance to which the partial decalcifying action of the bichromate of Zenker's and Müller's fluid (in which the dura was successively placed) contributed.

A microscopic cross-section of the tumor shows both the central stem and the processes which grow from it to have the structure of the lamellated bone. The ground substance is quite homogeneous, and only in places can the circular lamellæ be clearly made out. Between the lamellæ and scattered in the homogeneous osseous substance slit-like spaces are found, in which small nucleated bone corpuscles appear. There are no canals in the worm-like processes, and no Sharpey's fibers. The only exception to the homogeneous, delicately lamellated structure is found at the periphery of the central stem, which is bounded by a thin membrane composed of several layers of laterally compressed, spindle-shaped cells (imperfect periosteum?).

CLINICAL RÉSUMÉ.

So far as the tumor in the cranial dura is concerned, it probably was responsible for the epilepsy with which the patient suffered, and being of that class of neoplasms which grow deliberately, it accounts for the duration of the epileptic phenomena as stated in the history of the case. Situated so as to impinge upon the upper portion of the motor cerebral cortex, and ultimately making enough pressure to cause a distinct atrophy of the cortex beneath it, there can be no question as to the irritative influence of this foreign body upon the sensitive brain substance. Thus the case should be regarded as one of *secondary epilepsy*, in which a causative rôle was played by the dural tumor. From what we know of cases of this kind, the convulsions should have been Jacksonian, at least in their commencement, beginning in the toes and foot of the right side; but on this point the clinical history is not clear. Had such a history been obtained and controlled by careful examination and observation, no doubt a diagnosis of Jacksonian epilepsy might have been made, and the case could have been subjected

to surgical treatment, possibly with a favorable outcome so far as the dural growth and its irritative effects were concerned.

Nothing in the history as recorded points to the presence of the two other cerebral tumors—the cavernoma in the callosal gyrus and that in the optic thalamus. These tumors, like the one in the spinal cord, were doubtless of comparatively recent origin, or at least their final growth was rapid; and most likely the three neoplasms sprang into existence almost simultaneously, so that the most serious one, that in the spinal cord, giving the principal clinical symptoms, may have masked evidences of those in the brain.

As to the spinal tumor and its accompanying hemorrhage, it bears out the clinical symptoms referable to this region—those of a gradually progressing transverse destruction of the cord. Here the cavernous angioma, originating in the pia, was the primary lesion, in itself enough to cause considerable disturbance, but not enough to effect the complete section of the cord, which was later brought about by the massive hemorrhage. It is important to note the persistence and exaggeration of the deep reflexes, almost to the day death took place.

No clinical evidence of the hematomyelia was revealed, this being again a feature which was clouded by the more extensive transverse lesion in the cervical cord.

PATHOLOGICAL RÉSUMÉ.

Three distinct varieties of mesoblastic neoplasms were found in the post-mortem examination of the central nervous system of this case, and there is no reason to believe that these varieties were in any way related. The dural tumor belongs to a type quite common about the brain and spinal cord, and has no special claim upon our further attention.

An interesting point relative to the osteoma of the spinal arachnoid concerns the genesis of these tumors. Osteomata are not uncommonly met in connection with the cranial dura, especially about the falx, where they usually appear as osteophytes—irregular plates or spicules of bone, and owe their origin to the periosteal or bone-forming activity of the hard brain covering. Such formations are less common about the spinal dura, though they have been often described.

In the spinal arachnoid osteomata are also quite frequently found, though here they usually appear as osteophytic plates or spicules. In our case, however, the tumor has more claims to be called a neoplasm in the strict sense, for there is produced a mass of new osseous tissue of a peculiar form; this vermiform tumor appearing in the arachnoid. As a basis for the more extensive and peculiar portion of the tumor is a delicate spicule of bone, also lying in the arachnoid, but at one end *connected with the dura*. To this dural connection the development of the osteoma is probably to be charged, for as Zanda² has prettily shown, these tumors come about after this fashion: The primary departure towards the formation of a neoplasm takes place in the connective tissue of the arachnoid, which, in response to some irritation, probably originally inflammatory in nature, proliferates, but no formation of bone takes place so long as the newly formed tissue remains localized in the arachnoid. A sprout reaching the dura, however, this membrane assumes a periosteal function, bone is deposited, and thus the connective tissue new growth becomes an osseous neoplasm.

Some explanation of the anatomical diagnosis of the vascular tumors in the brain and cord may be desirable, for at the present time there is so strong a tendency to class almost all neoplasms arising in the substance of the central nervous system with the gliomata that some good reason for not doing so must be given. Besides, the appearance of three similar mesoblastic tumors in regions remote from each other at first suggests their metastatic origin from a primary malignant new growth, so that it becomes desirable to indicate why these particular tumors have been looked upon as multiple angiomata, and not as sarcomata with vascular proliferation.

In these cavernomata, and especially in that found in the optic thalamus, the presence of a network of neuroglia at the border of the blood-spaces and nervous tissue is noted, and unless the presence of this neuroglia can be otherwise explained it may well be concluded that a gliomatosis is the primary lesion and the blood-vascular phenomena are accompaniments, making the neoplasms *telangiectatic gliomata*. But the bulk of the tumor

²“Ueber die Entwicklung der Osteome der Arachnoid spinalis,” Beiträge zur Patholog. Anat., Bd. V., 1890.

is made up of communicating, endothelial-clad blood-spaces filled with blood elements, and here there is nothing that would make one hesitate to pronounce it a cavernous angioma. In the well-advanced tumors (callosal gyrus and spinal cord) a feltwork of glia fibers is only present at the periphery of the new growth, appearing as a wall or barrier between the neoplasm and the nervous tissue roundabout—as though new neuroglia arose (or that formerly present condensed) in response to the irritation of a foreign body, just as connective tissue responds to such a stimulant. It is this view of the matter that is held, although our knowledge of the regeneration of neuroglia is still meager, and the question of how this tissue reacts to foreign bodies far from settled. A circumscribed (and *tumor-like*) glia proliferation has, however, been described by Lubarsch³ in the case of an echinococcus cyst of the spinal dura, which effected a narrowing of the spinal canal and pressure upon the spinal cord.

On the whole, the phenomena witnessed in the neuroglia bordering these tumors in our case resembles that seen in the region about old hemorrhages, where the same large protoplasmic cells, pigment-containing cells, new blood-vessels, and increased neuroglia fibers are to be successively seen. Even here the origin of the peculiar cells appearing about the foreign blood mass has not been finally elucidated, as is forcibly put by Ribbert⁴ when he asks: "Welcher Art sind nun diese Zellen?" and then proceeds to answer the question in part, at least. The whole question of gliosis, and gliomatosis, becomes involved in this matter when it is fully probed, and it is foreign to the present purpose to make an elaborate discussion of these very interesting and important topics.⁵

Considering the possible sarcomatous nature of these neo-

³Ergebnisse der allgemeinen Pathologie. II Abth., 1895, p. 340.

⁴"Lehrbuch der Pathologischen Histologie," p. 401.

⁵A somewhat more complete presentation of this case will appear in the forthcoming number of the *Bulletin of the Ohio Hospital for Epileptics* (Vol. I, Nos. 2 and 3, July and December, 1898.) It is my desire, however, to give certain features suggested in the course of its study a special and more detailed setting forth, especially with reference to the part played by the neuroglia about the various neoplasms; and as to the comparison of the hematomyelia and syringomyelia, here again noting especially the deportment of the neuroglia. At this time the literature involved will be discussed.

plasms, the only suspicion that could be entertained would concern the pigment-containing cells in the perivascular spaces and scattered in the connective tissue and neuroglia about the tumors. But these cells are disposed in such a way as not to resemble sarcomatous cell-groups, and they are all so readily accounted for on the phagocytic basis that it seems hardly necessary to dwell on this point. Another argument against the possibility of these cells being sarcomatous elements is their presence about the blood-vessels and in the spinal tissue bordering the hematomyelia, even at a distance of fifteen centimeters from the spinal angioma.

Dismissing the suspicions of glioma and sarcoma, it only remains to repeat the conclusion already reached concerning the nature of these neoplasms, viz., that they are multiple cavernous hemangiomata, or cavernomata.

Cavernous hemangiomata are rare tumors in the central nervous system; they are specially uncommon as multiple tumors; so that the presence of these tumors gives this case a distinction, even without its other interesting features. Upon what supposition can the (probably) simultaneous appearance of these three cavernomata be best explained? Most satisfactorily this question can be answered by accepting the views of Ziegler and of Thoma relative to the origin of cavernous angioma (especially of the liver) in advanced life. Ziegler⁵ does not regard these formations as true tumors, but ascribes their inception to a varicose dilation of certain capillaries which afterwards communicate by an atrophy of their walls, a new formation of blood-vessels not taking place until after the cavernoma has become well advanced. These secondary angiomata differ materially from the congenital cavernomata of childhood, in which a true neoplasm forms. A further explanation of the production of these "senile" cavernous angioma is given by Thoma,⁶ who has especially studied the histo-mechanical phenomena and has shown that in the production of all blood-vascular tumors alterations in the blood pressure and the velocity of the blood flow play most important parts: according to which an increase in the blood pressure and in the velocity

⁵ "Lehrbuch der pathologischen Anatomie." Siebente Auflage, 1892.

⁶ "Lehrbuch der allgem. Pathologie," Bd. I.

tends to produce an angioma with dilated walls, as seen in cavernous angioma. By assuming the operation of such factors as these, the appearance of the multiple angiomata in our case can be more readily understood than upon any other hypothesis.

The massive hemorrhage in the enlarged cervical portion of the spinal cord is probably to be ascribed to the rupture of certain vascular channels composing the angioma which makes a part of this enlargement, and the blood-filled tubular canal which extends both above and below the cervical region is probably only a longitudinal extension of the same hemorrhage into the substance of the cord. In attempting to explain the occurrence of this hematomyelia the whole question of cavity formation (syringomyelia) in the cord is involved—a question not yet settled, and one that must here be avoided. One point only need be touched, and that refers to the absence of a pronounced gliosis about the spinal blood-tube; since this is the feature which distinguishes the lesion from an ordinary syringomyelia, though the writer inclines to believe that this is simply a question of time, and that, had a sufficient interval elapsed after the occurrence of this tubular spinal hemorrhage, a gliosis, reparative in its tendency, would have been found around the tube, just as it was found about the small neoplasm in the thalamus, and to a lessened degree about the larger and probably more actively-growing cavernomata in the callosal gyrus and in the spinal cord. In this light it is proper to regard the massive blood-tube in the enlarged cervical cord, and the smaller longitudinal tubular excavation (hematomyelia), as recent lesions of traumatic origin, and to them, especially to the massive clot in the cervical cord, the rapidly fatal progress of the case must be ascribed.

RETARDATION OF PAIN-SENSE IN LOCOMOTOR ATAXIA.

(PRELIMINARY COMMUNICATION.)

By L. J. J. MUSKENS, M.D.

ASSISTANT IN NEUROPATHOLOGY, CORNELL MEDICAL COLLEGE.

That *retardation* of the sensibility for pain is not at all rare in locomotor ataxia is now well known. Attention was called to this fact by Cruveilhier, Charcot, Leyden, Naunyn, Weir Mitchell, Gowers, E. Remak, Berger, Bolkostern, and M. Lähr. Most observers point out, also, that this retardation is often met with in the lower extremities; more detailed localization of this qualitative sensibility-disturbance I have not found.

In one case of undoubted and well developed tabes dorsalis I found a circumscribed circular area of analgesia below the knee, while the sense for touch was intact. Examining carefully the zone where the algesia went over into the analgesia, I found that the response of the patient on the sticking with a needle was usually delayed. He remarked also after inquiry that he felt two distinctly separated sensations, first the touch, then the pain. The same patient had, like many well developed cases, a broad zone of analgesia over the upper chest (this is also in incipient cases a rather frequent and early symptom of the disease), whereas his sensibility for touch was well preserved. On the borders of this area, as well as on those of the analgesic (ulnar) side of both arms, a similar condition was found almost constantly, viz.: *A zone of retardation of pain-sense on the border of the analgesic areas.*

This observation seemed remarkable enough to lead me to examine with particular interest the borders of the analgesic zone in all further cases, viz.: the edges of the analgesic area where the sense for touch was not disturbed. This was done in 31 cases. Examining with the necessary care and patience, I could find in all cases, at least on one or more of said localities, more or less retardation.

Quite an amount of power of observation and analysis of sensation is needed on the part of the patient to distinguish qualitatively different sensations; and it is necessary in almost all cases to help the patient in a way; to ask him which he feels

first, the touch of the pin point or the pain. If one examines a patient on the said localities, under said conditions, the patient will soon catch the meaning of the questions and answer either: "First the pain," or, "The touch and then the pain;" or, if the pin was pressed in the skin of the algesic zone: "First the pin," or, "Both together at the same time." This method has proved to me to be much more useful than that usually employed, when the psychical reaction time is observed, which is very different in different patients, and varies decidedly in less educated people.

That the border between algesic and analgesic areas often is found very different, even within a few minutes, and that there are many factors which must be regarded when locating this border, is a fact to be borne in mind. The zone of retardation varies very much in broadness, from a millimeter and less to some decimeters. Large zones one is apt to find more in the lower extremities, whereas the zone in many old cases of the disease cannot well be found on the upper border of the analgesic zone of the chest, and on the borders of the ulnar analgesic zone on the upper extremities. It appears as if the retarded zone with particular regularity is found where the analgesia is developing, and cannot so easily be found when the process of destruction of centripetal fibers has come to rest.

For several reasons I am led to believe that this retardation is an important and early symptom of the disease, and probably always precedes the total loss of sensibility for pain. This retardation is probably of differential diagnostic value. It seems to me that retardation of conduction of stimuli is a more frequent biological phenomenon than is ordinarily supposed. The retardation of the conduction of the contraction wave in the cardiac muscle under influence of the pneumogastric nerve was studied by me not long ago,¹ after Engelmann had observed the same phenomenon in the dying heart. As to the retardation in the centripetal nerve fibers in diseases of cord and peripheral nerves, it is quite clear that where the nerve fibers are slowly perishing, the first phenomenon may be re-

¹ L. J. J. Muskens, "An Analysis of the Action of the Vagus Nerve on the Heart." *American Journal of Physiology*. I, No. IV., 1898, p. 486.

tardation of the conduction of the impulses, and that only later the loss of sensibility for said quality may be total.

For the great clinical material necessary for this kind of research I am indebted to my chief, Prof. C. L. Dana, and to Prof. G. M. Hammond; for cases also to Dr. J. Fraenkel and Prof. J. Collins.

CONTRIBUTION TO THE SYMPTOMATOLOGY OF INTRACRANIAL DISEASE.*

By JOSEPH FRAENKEL, M. D.,

PHYSICIAN IN CHARGE OF THE MONTEFIORE HOME: INSTRUCTOR IN NERVOUS DISEASES AT THE CORNELL UNIVERSITY MEDICAL SCHOOL; NEUROLOGIST TO THE CITY HOSPITAL, NEW YORK.

Modern neurology is justly proud of the achievements in the recognition and management of intracranial affections, especially brain abscesses and tumors. But we must all concur in the opinion of Oppenheim, expressed at the late International Medical Congress at Moscow, that the diagnosis of these conditions is yet far from what it should be, and that the uncertainty of diagnosis is the cause of therapeutical, especially surgical, failures. So I venture to add to the abundance of clinical reports the history of two cases, if only for the sake of fortifying and supporting facts already known and established. The cases that I am about to report have given me much clinical and therapeutical information, and I therefore ask indulgence through the lengthy recital.

Case I. The patient, a boy seven and a half years old, was brought to the Montefiore Home, September 2, 1896, by his mother. She related in a husky voice and hurried language the history of her son's illness. The explanation of the mother's unusual behavior was readily apparent in her tremor, struma, and exophthalmus. She said that she was the only member of her family who was not well. Repeated interrogation failed to elicit the statement of disease of any kind in the little patient's ancestry, except the condition of the mother. The child was born at full term, grew and developed normally, had croup and measles in infancy. He attended school, and, so far as could be learned, was bright and normal in every respect until April, 1896. At this time, one day while at dinner he suddenly took the spoon with which he was feeding himself and shifted it from the right to the left hand and continued his meal. When asked the reason, he simply stated that he was unable to do it with the right. Soon afterward the mother noticed clumsiness of the right lower extremity, and the boy frequently asked to have removed from his shoe a nail which, he thought, impeded locomotion.

* This paper was read, and one of the patients and the specimen from the other were presented before the New York Neurological Society, January, 1898.

These symptoms continued for about three weeks; and after a stay of about two weeks at the Post-Graduate Hospital, the patient left considerably improved.

Two months before coming to the Montefiore Home, attacks of vomiting occurred and the patient complained of severe headache, and became dull and drowsy. The motor impairment of the right side grew worse, and a right facial palsy developed.

The mother claimed to have noticed twitchings, "grasping-like movements," of the left upper extremity, especially when the patient was asleep, and she stated further that he moved the right upper extremity when asleep in a way that he was unable to do when awake.

At no time was there any disturbance of speech noticed by the mother, and there was no incontinence of the bladder or rectum.

The mother was entirely ignorant of any cause for the onset of these symptoms, such as traumatism or acute febrile disease, and she did not notice any minor premonitions of the symptoms.

Examination. The patient was fairly well built, and weighed 41 pounds. The lower abdomen was somewhat protruding. The body showed a number of bruises, which were the results of frequent falls. The lower extremities showed a few pigmented, atrophic, cutaneous scars. Glands, especially in the groin and cervical region, were small, round, and hard. Head, asymmetrical, left parietal bone somewhat more highly arched. There was a slight convergent squint, and the ears were large. The incisor teeth were somewhat serrated, although they did not approach the so-called Hutchinson type, for they showed a convexity, not a concavity, in the middle, and the enamel was intact. Right angle of the mouth drooped. Right upper extremity was rotated outward at the shoulder, semiflexed and pronated at the elbow; fingers flexed at the second phalangeal joint, the thumb abducted. The right foot was incurvated. When the patient stood, the eyes open, there was distinct swaying, which increased when the eyes were closed, as did also the play of the tendons of the right foot (fork symptom). The gait may best be described as ataxic-hemiplegic. The deviation from a pure hemiplegic gait was that the patient put his right foot to the floor with varying force at different times, and frequently on top of the other foot.

Pulse 72, soft and small. Respiration 24; temperature 99°. Percussion of thorax showed nothing abnormal, but the note on the right side was of higher pitch than on the left. Respiratory murmurs; inspiration, as well as expiration, diminished on the right side. Heart dulness was a trifle increased to left side. The rhythm was slow, the sounds muffled.

The lower abdomen was bulging, and on palpation it conveyed to the finger the sensation of a cystic mass, situated in the middle line. This tumefaction was taken for a distended bladder, and patient was given a vessel and asked to pass urine. It took him more than fifteen minutes to start the stream, and he then passed about eight ounces of urine, whereupon the tumefaction disappeared. The urine was pale, of neutral reaction, specific gravity 1003, without abnormal constituents.

Nervous system. The patient showed considerable hebetude, although he answered questions correctly and did everything that he was told to do, but rather slowly, after long deliberation and in an automatic fashion. One could elicit no distinct defects of the intellectual or associative faculties, and there were especially no aphasic symptoms. Speech was slow and stammering. He wrote *very* poorly with the right hand, the letters being ataxic; and he did not write much better with the left hand.

Vision seemed somewhat impaired on both sides; there was no hemianopsia. Optic discs were not very clearly outlined, and there was distinct tumefaction of the right disc. The pupils were of medium size, unequal, the right wider. The reactions were prompt and alike on both sides. Although each eyeball could be moved freely in every direction, the conjugated excursions of the eyeballs were insufficiently balanced and accompanied by irregular rotatory and lateral nystagmus-like movements. The muscles mostly affected seemed to be the left external and right internal recti.

Smell and taste were unimpaired; hearing was distinctly impaired on the right side; the bone conduction on this side was absent. Both drum membranes were retracted, especially the right.

It was seen at a glance that the patient had an inequality of facial innervation. This was conspicuous when the countenance was in repose, and when it displayed spontaneous, unconjured emotions. On intentional and volitional innervation the two sides of the face responded equally. When the patient was told to laugh, *i. e.*, to make facial grimaces, the external equivalent of laughing, the asymmetry of the face was not seen, but at once became evident when the laugh was the externalization of a psychical equivalent. Moreover, it was to be remarked that the facial asymmetry was more conspicuous when the patient smiled than when he laughed aloud. If the smile was preparatory to hearty laughter, the unequal innervation of the face was first very manifest, and as the smile transformed into laughter it gradually became less evident and finally dis-

appeared, while the laughter continued in a spasmodic way for some time.

Innervation of the soft palate was equal on both sides, but the tongue apparently deviated to the right.

Percussion of the skull was not painful, and auscultation of the percussion did not yield any information.

The right upper extremity was flabby and flaccid, of smaller volume than the left, and passive movements encountered no resistance.

Right lower extremity showed similar abnormality; the large toe was in a state of hyperextension, and the foot showed considerable excavation of the plantar surface.

Motor power of the right upper extremity was diminished, and this was more evident when the co-ordinating functions were examined than by dynamometric examination. There was no tremor of the upper extremity.

The motor power of right lower extremity was hardly diminished; but it was very difficult for the patient to move the toes or right ankle joint properly and to the normal extent. Very distinct ataxia in the right lower extremity.

Electrical reactions of the muscles of the right upper and lower extremities were diminished only to a slight degree.

The superficial reflexes were lessened on the right side. Patellar jerk livelier on right side, and there was an easily exhaustible ankle-clonus on this side. Jaw-jerk was present.

Examination of sensibility was rather unsatisfactory, on account of the age and mental condition of the patient; but positively there was no disturbance of sensibility of any account.

The patient was seen by Dr. Dana in the afternoon of the day when this examination was made, and the diagnosis of a subcortical brain tumor was confirmed. It seemed justifiable to locate the lesion in or near the optic thalamus.

The next morning the patient complained of severe headache and vomited frequently. He was irritable and dull, showed distinct spasmodic laughter and crying, with ready transition from one state to the other. He had not passed urine for 24 hours, and the bowels were constipated. Temperature, taken in the right axilla, was 99°; in the left axilla, 98.2. Pulse, 60, slightly irregular; respiration 18, irregular.

The state of spasticity and of the reflexes of the right extremities was rather interesting. At one moment, especially when the patient's attention was not directed towards the extremities, or when one happened to strike a moment of complete cessation of innervation, the extremities were absolutely flaccid and no reflexes could be obtained. At another moment a very slight resistance to passive movements was encountered; patellar jerk was fairly alive, and an ankle-clonus was easily demonstrated.

The patient was put on mercurial inunctions (60 grains daily), and on increasing doses of iodide of potassium. Very soon afterward there was improvement; the headache became less frequent and less intense, and he vomited only rarely. He was more wakeful, active, and playful. The dexterity of the right extremities returned to a considerable extent; the optic discs became normal on both sides, and the ocular excursions were fuller and more steady, and there was only a slight insufficiency of the conjugated excursions towards the left side. After a further lapse of about three weeks all subjective, and most of the objective, symptoms disappeared.

When examined five months later the patient exhibited the following symptoms: Right angle of mouth slightly lower. There was some swaying with eyes closed; in walking the patient carried the right upper extremity slightly flexed and rotated inwardly. Right lower extremity was held rigid at knee-joint, and from time to time the patient stumbled with right toes. Patient had forgotten what he knew about numbers and what he learned in school; in fact, he was silly, laughed spasmodically on the slightest provocation, and did it so persistently that his teacher was obliged to dismiss him from school. The skull was slightly tender to percussion over the left parietal region. There was convergent strabismus; left internus was in a state of slight contracture. Pupillary reactions were perfect. When the patient was asked to follow the examiner's fingers with his eyes there was noticed:

1. A decided sluggishness of execution, the eyeballs moving very slowly towards the direction indicated.
2. When in lateral position a deviation of the eyeball in the vertical direction; for instance, when looking towards the right an overaction of the superior oblique, and towards the left an overaction of the inferior oblique occurred.
3. All ocular movements were accompanied by nystagmus-like movements.

When the head was not supported it drooped slightly. To voluntary innervation right and left facial nerve responded quite normally, but on talking, or in repose, or on emotional display, a paralysis of the lower branches of the right facial was evident with all the above mentioned specifications.

The right extremities were thin and flabby; the spasticity varying; disturbances of co-ordination were well marked; no sensory disturbances.

Function of bowels and bladder normal.

Case 2. M. B., man, 29 years old, a peddler, single, entered the Montefiore Home, August 20, 1895, on account of pulmonary tuberculosis. The patient was born of healthy parents, and had a good family history. In 1893 he had typhoid

fever; in 1894, while in South Africa, protracted diarrhea and fever kept him in bed for two months. He was temperate, a hard-working man, free from venereal infections. He considered himself healthy until the symptoms of the present disease appeared about three years ago.

The usual forerunners of tuberculous infection were soon followed by pyrosis, distress after eating, and edema of the legs. On examination the patient exhibited the symptoms of a moderate lesion of both apices, and indications of mild Bright's disease. He remained comparatively well until November, 1896, when a new set of symptoms developed.

Without any premonitory symptoms or obvious cause for the superadded illness, the patient suddenly experienced pain and paresthesias in the left half of the face, including the oral and nasal mucous membranes and the teeth on the left side. At the time of the examination he complained of slight cough; swelling of the legs; paresthesias on the left side of face; attacks of severe occipital headache, associated with dizziness and vomiting; uncertainty of locomotion, especially while turning around. He felt all the time like a drunken man; bit his tongue when eating or talking, and thought that his eyesight, especially of the left side, had become poorer.

Examination. Pulse 96°; resp. 24; temp. 97°. Urine: pale, acid; specific gravity, 1014; contained a considerable amount of albumin, hyaline and granular casts.

Patient was of large frame, but anemic, and rather poorly nourished. Edema around ankles. Abdomen and lower thoracic aperture very much distended and barrel-shaped.

Examination of the internal organs revealed lesions of both lungs. The liver was considerably enlarged, the spleen slightly; the body was covered with cold, cutaneous and periostitic abscesses in various stages of development or retrogression. The clinical picture was so complicated that I shall omit the details of the symptomatology, excepting those bearing on the subject of brain tumor.

Head: Skull asymmetrically built, was not tender to percussion, and on auscultation no abnormality was noticeable. The most prominent feature was the facial expression. The left half of the face seemed contracted. Depressions were noticed corresponding to the temporal and masseter muscles of the left side. The left eyelid drooped, and was the seat of a few small herpetic blisters. The eyeball was injected and the cornea cloudy.

On superficial inspection it appeared as if patient had a right-sided peripheral facial palsy.

Smell and taste were impaired on the left side. Vision was impaired on both sides, but optic discs appeared fairly normal.

Hearing was good, but the patient complained of subjective noises in the right ear. Pupils of medium size, well shaped, equal on both sides, and reacted promptly. Nystagmiform twitchings accompanied the ocular excursions in all directions, most marked, however, when the eyes were turned to the left or upwards.

When the patient was put through the usual tests to determine integrity of the facial nerve, the right nerve was found to be entirely normal, while in the distribution of the left a phenomenon was noticed that can best be compared to the ataxia and over-innervation frequently seen in tabic patients. The patient performed all motor functions with the left facial—closing and opening the eyes, showing the teeth, frowning, etc.—with unnecessary force; with an expenditure of energy that was extravagant in comparison with the effect desired.

He was unable to chew with the left side, and on swallowing he experienced slight difficulty with large pieces. Food frequently lodged on the left side, and he was unable to dislodge it in the usual way. The excursions of the lower jaw were limited, the patient being unable to open his mouth as wide as he used to, and when doing so the protuberantia mentalis deviated towards the right side, probably on account of weakness of the left pterygoid muscles. Temporal and masseter muscles were atrophied, their mechanical irritability was increased, and the electrical reactions markedly but only quantitatively diminished. The latter was true also of the left facial muscles.

Sensibility of face was subjectively and objectively disturbed, and the patient himself mapped out the course of the first and second branches of the trigeminal nerve by indicating the situation of the paresthesia.

The objective sensory findings are shown by the accompanying figure. The sensory disturbances included the oral and nasal mucous membranes, and the left half of the tongue and conjunctiva. The tongue was dried and furred, particularly the left half. It protruded straight, showed no tremor, and no inequality of volume. Innervation of the soft palate was normal, and motility and sensibility of the pharynx and larynx seemed undisturbed. Motility and sensibility of the upper and lower extremities were normal; the knee-jerks were very much diminished; there was slight Romberg symptom, but no evidence of ataxia, and the gait was slightly cerebellar.

The patient was ordered mercurial inunctions—75 grains daily—and increasing doses of iodide of potassium.

When examined after two weeks, the patient complained of severe left-sided headache, coming in attacks and lasting from $\frac{1}{2}$ to $1\frac{1}{2}$ hours. Vertigo and dizziness were very severe, so much so that the patient was unable to be out of bed. Appetite and bowels were fairly normal; micturition frequent.

The gait was uncertain, swaying, typically cerebellar. Romberg sign was distinct. The knee-jerks were very much diminished. Head and cranial nerves as before.

Treatment was continued for three weeks longer, and at the end of this time most of the symptoms disappeared; headache and vomiting ceased entirely, locomotion was undis-



FIG. I. *n*, area of absolute anesthesia; *o*, area of tactile anesthesia; *P*, area of paresthesia.

turbed, knee-jerks lively, sensibility of left side considerably improved.

He was seen by Dr. Sachs on two occasions, and the diagnosis of a lesion, meningitis or tumor at the base of the brain in the posterior fossa at the angle between the pons, oblongata and cerebellum was thought to be the correct one.

Unfortunately, the pulmonary and renal lesions were less amenable to treatment, and the patient finally succumbed in uremic coma; but at no other time were any brain symptoms noticed.

The post-mortem examination was made by F. W. Vissman, to whom I am indebted for the following notes:

Spinal cord showed no abnormalities; the arachnoid was

very slightly edematous in the upper cervical portions. The dura mater of the brain showed no abnormality; the arachnoid was slightly edematous over the vertex, and at the base it contained a number of gray, white nodules smaller than a pinhead. The lateral ventricles were of normal size and content. The ependyma was smooth, glistening and transparent. The vessels at base were thin and free from disease.

The pericardium contained about two ounces of a clear, straw-colored fluid; the walls of the sac were smooth and glistening, and there were no thickened spots. The heart itself was about the size of the fist of the corpse; the muscle structure was very flabby, and of a grayish-reddish color. The valves were soft and pliable, not thickened.

Parietal and visceral pleuræ were firmly adherent to one another over nearly the entire surface of the lung. On dissecting the apex a cavity of the size of a pigeon's egg was found, containing a creamy white liquid. There were a number of other cavities in the lungs, which varied in size from those described down to those of the size of a cherry. The walls of these cavities were rather clean. In other portions of the lung we found the air cells filled with dark, red blood, which gave the cut surface a rough, velvety appearance. In other places the air cells were dilated, but contained air. The right lung showed practically the same changes as the left lung, excepting that it contained no cavity with pus.

The spleen was enlarged and very firm; its color was dark red; on the cut surface numerous glassy-looking spots about the size of a hempseed were visible. Capsules smooth and transparent.

The left kidney was large and grayish-yellow in color. Its surface was very slightly mottled and smooth. The cut surface showed the cortical portion thick and very slightly prominent above the medullary portions, and very pale. In right kidney the same conditions were found as in the left.

The liver was large, the edges round, the color was grayish-red, consistency very firm. The cut surface appeared cloudy. The central veins and the lobule were not easily distinguished with the naked eye.

Immediately below the left kidney a tumor of the size and shape of a goose-egg was found. This tumor seemed to be attached by a pedicle to the left side of the bodies of the fourth and fifth lumbar vertebræ. From there it extended down about $2\frac{1}{2}$ to 3 inches in the psoas muscle. On removing the tumor it was accidentally cut and a creamy, white fluid flowed out. The inner surface of the cyst was smooth, except where it emanated from the last lumbar vertebra: there was a rough erosion, which admitted the end of the little finger.

Diagnosis: Meningitis basilaris tuberculosa; Pleuritis ad-hæsiva chron.; Phthisis pulmonum; Hydrops pericardi; Hypertrophia lienis; Degeneratio-amyloidea lienis; Hepatitis parenchymatosa et interstitialis chron.; Caries ossis vertebræ lumbalis V.

I will add that there was some dispute at the post-mortem table about the existence of any intracranial lesion at all. To me the pia mater at the base of the brain in the left ponto-medullo-cerebellar angle seemed thickened, and a few white, rough streaks were seen. But my belief was not shared by the others present. On the contrary, the opinion was expressed that the whole symptom-complex was a functional one, and probably caused by the renal affection—uremic in nature.

Twenty-four hours afterwards, when the specimens were looked at again after a stay in Müller's fluid, a unanimity of opinion that the pia was abnormal was reached. In addition a few small nodules of the size of a pinhead were noticed on the specimens.

A review of the symptoms of these two cases necessitates further comment:

The diagnosis of an organic, intracranial lesion, in fact of a brain tumor, was positive in the first case, but whether the nervous symptom-complex of the second case could not just as well be explained by the assumption of a functional, say uremic toxemia, as was suggested at the autopsy table, had to be carefully considered.

Of course, the constancy and the clearness of the clinical picture, the absence of other uremic symptoms, and the presence of some undoubtedly organic symptoms, pointed rather strongly towards the organic nature of the disease, tumor or meningitis.

The positive discrimination between a solid tumor or localized basal meningitis was in this case, and is often, almost impossible. During life the absence of a stiff neck and of permanent headache were more in favor of the diagnosis tumor, and from the post-mortem findings the limited localization, the normal appearance of the rest of the meninges, seem to point in the same direction. In determining the nature of the lesion the age of the respective patients was somewhat misleading.

Following the teachings of text-books, such as Gowers', Dana's, Sachs', Starr's, Dercum's, Oppenheim's, and others, in which rarity of gummatous lesions in children

and prevalence of such in adults is strongly emphasized, one would at first feel inclined to regard the lesion in the first case as a tubercle, and to suppose a specific infection in the second. But the pulmonary affection of the second patient (although tubercle bacilli were never found in the sputum despite repeated examination); and the swollen glands, the formation of the teeth, and the cutaneous scars in the first case, equivocal as their interpretation may be, were of considerable aid in the decision. The remissions and the interrupted course of the disease in the first case were of great value in deciding in favor of a specific lesion.

The general symptoms of intracranial affections were present in both cases; in the first more marked than in the second; the absence of optic neuritis in the second case is the more remarkable, as this symptom is said to be very common in affections of the posterior fossa.

The special and localizing symptoms of the second case presented a fairly clear ensemble, characteristic of lesions of this part of the brain. Whereas, the disease of the fifth nerve made the basal and superficial position of the lesion highly probable; the purely cerebellar symptoms of dizziness, cerebellar gait, and especially diminution of the knee-jerks, were perhaps too strongly outspoken for a lesion at this situation, a lesion that presumably was not very large, as the absence of disease of other nerves of the base of the brain, and especially the absence of bulbar symptoms, seemed to indicate.

In this connection it is well to call attention to the symptom pointed out by Stieglitz as being of great value in the initial diagnosis of cerebellar lesions; I mean the diminution of the electrical response of the facial nerve, which was present in our case.

Affection of the motor portion of the trigeminal is a rather uncommon feature of trunk lesions of this nerve. Leube states that in trunk lesions of the fifth nerve the sensory fibers are frequently the only ones to suffer, the initial irritation (neuralgia) being soon followed by paralysis (anesthesia).

Concerning the lesion of the fifth nerve, especially of its motor portion, the clinical evidence of a disease of the pterygoid muscles deserves special mention. The limitation of

the excursions of the left mandibular joint and the deviation of the lower jaw towards the healthy side could hardly be explained on any other basis. The symptomatology of the lesion of the sensory branches shows in our case a feature rarely described: the above-mentioned over-innervation of the facial muscles—let us call it the facial ataxia.

And, in fact, one often notices in tabic patients an undue widening of the palpebral fissure, alternating with a slight ptosis. Every time that I have found this symptom there has been disease of the fifth nerve.

It brings forcibly to one's mind the belief that a great deal of the purely ataxic symptoms observed in other cases, especially in cases of tabes, is conditioned by an interference with sensory, or, better said, centripetal impressions.

The occurrence of trophic disturbances—herpes, injection of the eyeball, and cloudiness of the cornea—is not entirely in accord with recent physiological teaching, explaining these symptoms by the assumption of a nuclear lesion or a disease of the Gasserian ganglion.

We are not justified from the post-mortem findings in venturing an explanation of the diminished reflexes; and to speculate about it seems unwise and untimely when new theories about this subject are brought out almost daily.

The symptomatology of the first case was very striking, and, in view of the scarcity of cases of this type, seems to deserve a more detailed consideration.

Cases of tumor of the optic thalamus have been reported by Nothnagel, Dejerine, Dana, Oppenheim, Dercum, Sinkler, and others; but in most of them the lesion was rather extensive, and it is somewhat difficult to enucleate the clear picture of thalamic affection.

Before going further, it seems suitable to recapitulate briefly the anatomy and physiology of this part of the nervous system. Our knowledge in this direction is meagre indeed, but only recently a noteworthy addition has been made to it by Exner, Bechterew, Simbriger, and others. Frequently the statement is made that disease of the basal ganglia rarely gives rise to any special localizing symptoms throughout the whole course when general symptoms are fully developed, and in the last

edition of the "Traité de Médecine" (Vol VI.), for instance, one finds the statement concerning tumors of the basal ganglia: "Celles-ci ont une symptomatologie beaucoup plus confuse encore. Non seulement elles n'ont rien de pathognomonique, mais elles peuvent évoluer sans symptômes. Leurs symptômes, lorsqu'elles en ont, échappent à toute combinaison nosographique. Seuls, les symptômes des tumeurs, en général, existent."

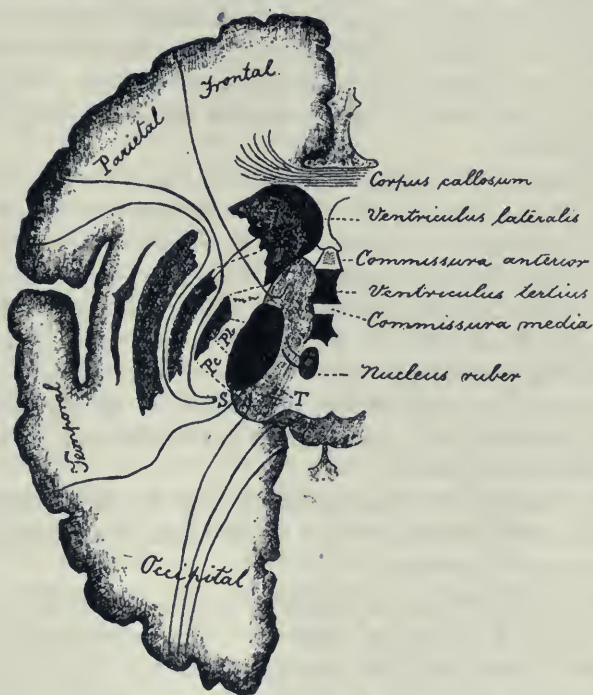


FIG. II. T, probable situation of tumor; m n, pathway for motor cranial nerves; Pb, pyramidal fibers for upper extremity; Pc, pyramidal fibers for lower extremity; carrefour sensitif.

The accompanying cut, modified after Leube, gives a clear conception of the position, surroundings, and intracranial connections of the optic thalamus that are so far known.

In a recent article in the *Neurologisches Centralblatt*, Bechterew claims to have found the centripetal and centrifugal thalamo-spinal connections. The former are supposed to be represented in fibers of the posterior columns, the latter to run

down with the pyramidal tracts, situated in the internal capsule, in front of the bundle representing these fibers here.

In the same article Bechterew calls the optic thalamus a center for the complicated psychical reflexes, a center regulating the expressions of the emotional state and the reflex functions of the vegetative organs. This author is in agreement with Exner, Starr, Dana, and other writers on the subject.

Taking it altogether, it seems that the optic thalamus is a center regulating and governing the functions midway between intellect and instinct; functions which by ancestral or personal habitual performance have become more or less permanently organized, and are generally executed without interference of consciousness.

In general, the symptomatology of the first case coincides fully with the classical descriptions characteristic of lesions of the optic thalamus. Nearly all symptoms that are mentioned in this connection were present in this case: emotional spasticity, mental hebetude, pupillary and ocular symptoms, abolition of the psychical reflex of the seventh nerve, mild and slowly progressing hemiplegia with inconstant spasticity, muscular atrophy and inco-ordination, depression of all vegetative reflexes, were and are in part present yet in this case, as in other cases hitherto reported.

The change of the percussion note of the right thorax and diminution of respiratory vigor of the viscus is a symptom that has not been described.

It is rather difficult to imagine that this occurrence is a part of the general vegetative depression, the one-sidedness being an almost insurmountable obstacle for such a conception. It would satisfy one more to assume that the change in percussion and auscultation was produced by a physical cause—the flabbiness of the resonating walls of this side. From the symptoms enumerated it is difficult to choose the ones or one that could be regarded as the direct focal symptoms.

In looking at the accompanying diagram and imagining the supposed tumor to be situated in the anterior part of the optic thalamus, the ocular and hemiplegic symptoms could be explained by pressure on the capsular pathways for the cranial motor nerves and the motor fibers for the opposite side

of the body. It would be equally well to look for an explanation of the inco-ordination in the same direction. Widening of the pupil and the lowering of temperature on the paralyzed side are frequently mentioned as thalamic symptoms, but are, unfortunately, very inconstant. The muscular atrophy, the general depression of nutrition and vegetation, are, however, no common associates of capsular hemiplegias. Uniformly the paralysis of the psychical reflex, expressed through the facial nerve, is looked upon as the characteristic thalamic symptom. The pathomechanical explanation of this symptom is entirely hypothetical. In spite of this, I would join the majority and look at the symptom as a pathognomonic one, were it not for a recent experience which makes me cautious of accepting this view unconditionally. I refer to a case of undoubted peripheral palsy of the seventh nerve that I had occasion to observe some time ago. After the improvement and disappearance of most of the symptoms, the symptom that persisted permanently was mimetic palsy. The case altogether was a rather uncommon one, inasmuch as the facial palsy was the first expression of constitutional syphilis, and it preceded the appearance of the cutaneous symptoms by several days. The roseola was to be noticed five days after the onset of the sudden facial palsy. Whereas the electrical reactions and the intentional innervation of the face muscles returned rather quickly after specific medication, the interruption of the physical reflex is still noticeable in the patient. In attempting to explain this phenomenon, bringing in the optic thalamus was manifestly out of the question. The unusual etiology in this case, being more malignant than the common cause (exposure to cold), is, perhaps, evidenced in the clinical behavior of the affection. The syphilitic poison may either have altered the structures of fibers to such an extent that they were still able to convey volitional and electrical stimuli, but not subtle psychical impulses.

It goes without saying that this argumentation does not hold good for our case. I quote this experience, not to create further confusion, but to emphasize a general clinical rule, the special application of which to our case will prove its value.

Briefly, then, this rule says that the clinical and diagnostic importance of a symptom does not depend so much upon the

symptom *pes se* as upon its attributes, its character, color, tint, as it were, and its relations towards the other symptoms and to the general picture.

A symptom separated from its clinical company, or not sufficiently analyzed into its elements, loses a good deal of its value and is misleading in the final diagnostic calculations.

Now we return to our case and make application of the foregoing principle after again reviewing the symptomatology from this standpoint. The patient presented a hemiplegia, but not a hemiplegia in the usual sense of the word; a hemiplegia with very little paralysis of motor power, but pronounced paralysis of co-ordination—paralysis of a function that is either primarily instinctive or becomes so through habit. The defective ocular excursions may be looked upon in the same way as an inco-ordination of the external eye muscles. The mimetic palsy illustrates best the type of this paralysis, and lends a part of its clinical character to all the other symptoms. It is a paralysis of a purely instinctive motion, in this particular instance freed from obscuring combinations that prevent the thorough understanding and proper interpretation of the paralytic symptoms, in the eyes or extremities. The retardation of the general psychical functions, the retardation of the vesical and intestinal motility, tally very well with this conception, too.

What is, then, the pathognomonic symptom, or, better, complex of symptoms, of a localized lesion of the optic thalamus? Let us attempt to construct it.

From a review of the literature and an analysis of the case described, we would be justified in saying that a lesion of the anterior part of the optic thalamus gives rise to evidences of irritation or paralysis of the fibers situated near by, *i. e.*, the fibers of the anterior limb of the internal capsule. These symptoms are mostly or exclusively apparent on instinctive, habitual, and vegetative reflexes, the type being presented in the mimetic paralysis of the facial.

Lesion of the posterior half of the thalamus is frequently the cause of hemianopsia and other sensory disturbances. Whether an analogy could be established between the symptomatology of affections of the anterior and the posterior half of the optic thalamus future study of cases will have to decide.

Finally, let us consider the exact localization of the lesion in our case. The absence of hemianopsia or sensory disturbances on the opposite side of the body, the interference with the fibers for the cranial nerves in the capsular pathways, make it probable that the lesion was laterally in the anterior and middle portions of the optic thalamus (see figure).

The course and response to medication of our first case is a familiar occurrence. The persistence of some symptoms that are present to-day will have to be explained by the assumption that the previous lesion had destroyed some of the gray matter of the thalamus, and this destruction is like disease of gray matter in any other part of the nervous system—irreparable; or, on the other hand, by the supposition that the tumor is only arrested in its growth and somewhat shrunken.

The termination of the second case deserves a few words. The lesion was undoubtedly a tuberculous one and has, as the inspection of the specimen proves, almost entirely disappeared. The specimen further shows that no incapsulation or calcification has taken place.

With due regard to the *post hoc* fallacy, I am aware of the fact that spontaneous cures of tuberculous lesions have frequently been observed; though close relation in time between cause and effect, between specific medication and the disappearance of the clinical symptoms of the lesion, merits some consideration.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

May 2, 1899.

The President, Dr. Frederick Peterson, in the chair.

A CASE OF CHARCOT-MARIE-TOOTH AMYOTROPHY.

Dr. Frederick Peterson presented, on behalf of Dr. W. H. Caswell, a young girl who, when seven years of age, had an attack of measles. About two years later she became lame in one foot, and a few years later the other foot became similarly affected. Six or eight years after this atrophy began to develop, first in one hand, and then in the other. For a time the case was treated, as most of these cases are, as poliomyelitis. The history was characteristic, the speaker said, of a Charcot-Marie-Tooth amyotrophy. The picture presented was that of an old multiple neuritis. There was great wasting of the muscles of the extremities, including the smaller muscles of the hands. The peculiarity of this form of muscular atrophy was that it affected the legs below the knees, and the arms below the elbows, so that the limbs tapered off in such a singular manner that the diagnosis could be made from this aspect alone. The muscles of the roots of the thighs and of the trunk, on the other hand, were perfectly preserved. While there was no ataxia of the limbs, weakness made it impossible for her to stand still and she kept up a continual tramping, reminding one of the tramping of a horse in his stall. Reaction of degeneration in the muscles had been noticed for at least two years past. The knee-jerks were absent. The speaker said that the affection seemed to be a form of chronic progressive motor neuritis. It was a very rare disorder.

A CASE OF MULTIPLE TUMORS OF THE BRAIN, WITH AUTOPSY.

Dr. E. D. Fisher reported this case. The patient was a colored man, forty years of age, with a history of syphilis and alcoholism. He had been able to continue his occupation of cook up to about two weeks before coming under observation. When first seen he was weak on the right side, and when walking staggered toward that side. Examination had shown partial paralysis and partial anesthesia of the right side of the body. It was difficult to say at the time which side of

the face was paralyzed. Dr. Fisher thought he had been able to detect an incomplete ptosis of the right eye, which was partially overcome by drawing up the muscles of the forehead. This would partly explain the appearance of a partial paralysis on the left side of the face. Considerable exaggeration of the reflexes existed on both sides, and marked optic neuritis was detected. Under iodide of potassium the patient had improved decidedly, but soon afterward he had become almost completely unconscious. After a time he had recovered from the unconsciousness and had been able to go about the ward again. Soon afterward he had developed pneumonia, from which he died.

With such rather vague symptoms a positive opinion as to the location of the growth had been difficult. While in the hospital the patient had had two apparently typical epileptic seizures. The probable diagnosis had been a growth in the left side of the brain, in the region of the crura, or in the upper portion of the pons. At the autopsy a tumor, which was about half an inch in diameter, had been found in the left side of the brain, involving the lenticular nucleus on that side. This would have been sufficient to explain the partial hemiplegia. A similar growth was found on the opposite side of the brain, involving especially the optic thalamus. Microscopic examination by Dr. E. K. Dunham had shown the growths to be gummata. The post-mortem examination had also revealed multiple lesions in the liver and spleen. The spinal cord was not involved.

Dr. G. M. Hammond asked what were the symptoms attributable to the lesion in the thalamus.

Dr. Fisher replied that there had been no symptoms pointing to such a lesion, and it was this that had led to the erroneous diagnosis.

Dr. Fraenkel said that last year he had reported to this society a tumor of the optic thalamus, and had taken occasion to point out certain symptoms that he thought should be carefully looked for. It was generally stated that thalamic tumors usually did not give noticeable symptoms. It was not uncommon in the clinical examination to neglect to examine the facial musculature as carefully as other parts of the body. One reflex of the facial was the psychical or mental reflex. The behavior of the patient when laughing or crying was often significant in cases of lesions of the thalamus. These patients were able to make the grimaces expressive of laughter when told to do so, but the side of the face opposite to the lesion in the thalamus would not participate in the act of either laughing or crying. The patient in the case just reported probably had paralysis on both sides of the face—on one side a mental paralysis. Nothnagel had first pointed out this mental or psychical reflex. The behavior of the vegetative functions should be carefully watched in these cases, for there might be a transient paralysis of these functions.

Dr. Fisher said that the patient was a perfect mimic, and was laughing most of the time, and there could be no question that both sides of his face laughed, although there was always this difference be-

tween the two sides. Several physicians besides himself had watched this case, but had not agreed as to which side of the face was paralyzed. During life the case had suggested multiple tumors, but the location of the secondary tumor had not been exactly determined before death.

Dr. Noyes presented a man who had been injured last November by his right arm being caught in some belting. When he was seen in January there had been partial paralysis and anesthesia of the forearm. On February 1 the anesthesia was confined to the ulnar and posterior interosseous. Quite recently recovery had been fairly complete. A skiagraph was exhibited showing that callus had formed between the bones of the forearm. Pronation and supination were in abeyance. An operation would be necessary to restore the motion of the forearm, and the question arose as to whether such an operation would greatly interfere with the innervation of the arm.

Dr. Fisher said that the man had improved so much that he would suggest that the operation be postponed to allow of further improvement.

Dr. Noyes said that at first he had supposed the condition of the nerves was the result of pressure from the callus, but the rapid improvement had led him to think that it was possible that the injury to the nerves had resulted rather from wearing the plaster-of-Paris dressing than from the original injury. He was afraid that operation might lead to further involvement of the posterior interosseous.

Dr. Peterson said that from the history he would say that the case was probably one of paralysis from the pressure of the bandage or splint. With ordinary care the surgeon should be able to operate without inflicting further injury upon the nerves.

SENSORY DISTURBANCES IN EPILEPSY AND HYSTERIA.

Dr. E. D. Fisher read this paper, which was based on two cases, one of epilepsy and the other of hysteria, in which he had noted a peculiar distribution of the anesthesia. Richter had collected, he said, 71 cases of hysteria which had been examined especially as to the sensory disturbances. Dr. Fisher's patients had both been entirely unaware of their anesthesia until the examination.

The first case was that of a woman of seventeen years, who had had her first epileptic seizure at the time of the establishment of the menses. The seizures had recurred with each menstrual period, and without any aura. There was absolute anesthesia of certain parts of the face, trunk and upper extremity. Between the seizures the patient had been in full possession of her faculties. While there was a tendency for the anesthetic area to decrease, certain features had remained permanent. There was absolute loss of sensation over the central portion of the forehead from the glabella to the root of the hair. All portions of the scalp and face possessed normal sen-

sation. In the first two weeks there was some anesthesia over the shoulders and chest. The extensor side of the forearm and the dorsal surface of the hand exhibited a similar condition. There were no other symptoms indicative of disease.

The second case was that of a woman of twenty-four years, large and well nourished. She came to the hospital complaining of great pain on the left side, directly under the scapula. This pain was not increased by motion. Further examination showed plaques over the chest and shoulders which were absolutely anesthetic to all forms of irritation, and varied from time to time. The extensor side of the forearm and the dorsal side of the arm remained at all times absolutely anesthetic. The patient had remained under observation for several weeks, and died of pneumonia involving the side primarily affected.

The speaker said that these cases resembled in some respects examples of syringomyelia, and he was inclined to think that there was some spinal disturbance. One interesting feature was the similarity of the distribution of the anesthesia in cases of such different character, and coming under observation at the same time.

Dr. G. M. Hammond said that it had never seemed to him that the anesthesia was a symptom that properly belonged to epilepsy. The fact that the patient did not know of the existence of the anesthesia was not of much importance, for this ignorance was frequently observed in true hysteria. In many cases of epilepsy, particularly where the lesion was cortical and situated in the sensory areas, it was probable that there would be disorders of sensibility; nevertheless, he did not think such disorders should be considered pathognomonic of the epileptic state. Where such sensory disorders were observed he would rather believe that there was a hysterical element combined with the epilepsy. He would like to know if tests had been made regarding constriction of the color field and visual field, and a search made for the stigmata of hysteria.

Dr. Fisher replied that such an examination had been made in only one of the cases.

Dr. Fraenkel said that these cases only served to emphasize the importance of making careful examinations of the sensory disturbances. Such disturbances he knew personally varied greatly from time to time. He had made it a rule to have a chart made of the sensory conditions of every patient entering the Montefiore Home, and in studying these he had been astonished to find sensory disturbances in connection with tuberculosis and all sorts of diseases in which there was no disorder of the nervous system. Frequently they could be traced to slight injuries of the nerve filaments. The most interesting feature, however, was the variability of these disturbances. He recalled one case of syringomyelia that had been in the Montefiore Home for a number of years, in which he had discovered by accident a marked change in the sensory symptoms from morning to afternoon. He had not yet met with any cases of epilepsy presenting marked areas of anesthesia.

Dr. S. E. Jelliffe asked whether Dr. Fisher's patient had been taking large doses of the bromides, for in such patients there were

certain forms of anesthesia. The anesthesia of the pharynx was well known, and there were other anesthetics in other parts of the body observed in persons taking bromides.

Dr. Bailey said that he had been especially interested in the absence of all knowledge of the anesthesia on the part of the patient. It was important to draw the line between the functional and organic anesthetics, and it could often be done by this very question of the knowledge or ignorance of the patient of the existence of such anesthesia. He had never seen anesthesia extending down on the arms and hands, and resulting from organic disease, that the patient was not aware of. In typical hysterical cases the patients were not generally cognizant of its existence.

Dr. Peterson said that one should be very careful in studying the areas of anesthesia, especially in functional cases. He had observed marked variations in cases of syringomyelia, particularly in the boundaries of these areas. All were familiar with the transitory anesthetics occurring in epilepsy. Such instances, he thought, could be classified with the exhaustion pareses. In regard to the theory offered by Dr. Fisher as to the irregular areas of anesthesia distributed over the whole body in hysteria and epilepsy, he would say that he would look upon them as of a hysterical nature, and not of spinal origin. It was known that a hysterical element was present in epilepsy, as it was present also in tuberculosis and certain other diseases which influenced the nervous system quite profoundly.

Dr. Fisher, in closing, said that the two cases reported had been a surprise to him. They had presented exactly the same symptoms, and so far as he knew, these areas had remained unchanged. There was nothing remarkable about the anesthesia itself, or its variability. Anesthetics changing from time to time he certainly would not refer to the spinal cord, but when they were permanent it would seem that they must have an organic lesion as a basis. He was not speaking of these two cases alone, but of a number of similar ones reported by others. He had seen a case of syringomyelia in which there had been an area of anesthesia on the hand, and the patient had been entirely ignorant of its existence until his hand had been injured. One of the patients had never taken bromide, and the other had taken it in very moderate doses, if at all. He had never seen even the largest dose of bromide produce an anesthesia in which there would be no response under severe irritation. He felt sure that a great many more cases of this kind would be found, now that attention had been directed to the subject.

Dr. Bailey remarked that the case of syringomyelia referred to was hardly a fair one to cite as a proof of the existence of complete anesthesia without the knowledge of the patient.

Periscope.

ANATOMY AND PHYSIOLOGY.

124. UNTERSUCHUNGEN ÜBER DIE ANATOMIE UND PATHOLOGIE DES UNTERSTEN RÜCKENMARKSABSCHNITTES (Investigations on the Anatomy and Pathology of the Lowest Portion of the Spinal Cord). L. R. Müller (Deutsche Zeitschrift für Nervenheilkunde, 14, 1898, 2, p. 1).

Müller has made a careful study of the anatomy and pathology of the conus in man. He gives a summary of his most important findings. The anterior roots are much less numerous than the posterior in the conus, and the large motor ganglion cells are absent from the anterior horns below the third sacral segment; instead of these, groups of multipolar ganglion cells are found in the intermediate gray matter between the anterior and posterior horns. Fibers radiate in the conus from the posterior columns into the gray matter, and the posterior gray commissure is absent below the second sacral segment. The crossed pyramidal tracts do not extend below the third sacral segment; below this level the lateral columns are filled with fibers of other origin. Numerous fibers from the ganglion cells in the intermediate gray matter of the conus pass into the posterior part of the lateral columns, and fibers from the lateral columns pass into the posterior roots. The histology of the conus differs essentially from that of the remainder of the cord. Spinal ganglia belonging to the posterior roots of the conus may occasionally be found *within* the dura, and the lowest posterior roots not infrequently contain isolated spinal-ganglion cells. The posterior columns disappear in the lower part of the conus before the other columns disappear, and the central canal becomes enlarged and occupies the area of the posterior columns. The posterior columns in the lower part of the cord contain mostly centrifugal fibers, but the oval field of the lumbar region, the dorso-medial sacral bundle, and the triangular zone, degenerate differently in descending degeneration of the posterior columns. Descending degeneration in the posterior columns of the lumbar and sacral regions occurs only after an almost complete transverse lesion, and seldom extends below the third sacral segment, and the triangular field remains almost always intact. Descending degeneration is never found in the lateral columns in the lower sacral region. The degeneration in the posterior columns of the conus in tabes and cauda-equina lesions is slight. One of Müller's cases shows that a primary acute inflammatory affection of the cauda may occur, and cause the same symptoms as conus lesions.

SPILLER.

125. ZUR ANATOMIE UND PHYSIOLOGIE DES UNTEREN SCHEITELLAP-
SCHENS (The Anatomy and Physiology of the Inferior Parietal
Lobes). C. von Monakow (Archiv f. Psychiatrie, Vol. 31, 1898-99,
p. 1).

This is a paper of some seventy pages, and presents the author's preliminary contribution to the subject of the localization of the function of the gyrus angularis and the gyrus supramarginalis, with the anatomy of these parts of the parietal lobes. A number of observa-

tions on fetal brains and on experimental work with monkeys' brains are recorded, which admit of abstracting only with difficulty. With respect to the anatomy of these two portions of the brain the author states that as early as the fourth month ($3\frac{1}{2}$) it is evident that fibers arise in these two lobes which contribute to the formation of the corona radiata, the evidence for this being, in the author's opinion, even stronger than it is for the occipital lobes, *i. e.* the optic radiations. At this stage fibers are to be traced from the temporal lobes also. In his investigations on monkeys he was unable to actually demonstrate the relationship of these lobes to the corpora quadrigemina, but in some pathological human material he shows that destruction of the gyrus angularis leads to a degeneration of the anterior corpus quadrigeminum of the same side, and in a similar manner destruction of the anterior corpora quadrigeminal regions results in an ascending degeneration to the gyrus angularis. From one of these pathological cases it seemed evident that, following a hemorrhagic destruction of the pulvinar, seven months old, the dorso-lateral part of the lateral geniculate nucleus, as well as the posterior parts of the central nuclei groups, a secondary degeneration could be traced through the posterior internal capsule and extended in part through the optic radiations to the inferior parietal lobes. The continuation of the paper has not yet appeared.

JELLIFFE.

CLINICAL NEUROLOGY.

126. ZUR AETIOLOGIE DER ACUTEN POLIOMYELITIS (On the Etiology of Acute Poliomyelitis). Schultze (Münchener medicinische Wochenschrift, 1898, No. 38, S. 1197).

As a contribution to this question, the author reports the following: A boy of 5 years was taken ill with fever and slight vomiting, and two days later showed a paralysis of both arms and weakness of the neck muscles. Five days later he was free from fever, and on examination showed flaccid paralysis of both arms and of the neck muscles, with patellar and ankle reflexes lost on the left and weak on the right; both arm reflexes lost, other reflexes normal. There was no stiffness of the neck or back, and no hyperesthesia, although there was slight pain on pressure over the cervical spines. There was some somnolence for several days, but never coma. A lumbar puncture made 13 days after the beginning of the attack caused the escape under a pressure of from 500 mm. at the start to 200 mm. at the finish of 30 c.c. of a clear fluid containing at the end a few flocculi. The bacteriological examination of this fluid showed the presence of a diplococcus having all the morphological characters of the Weichselbaum-Jäger meningococcus, but no growth could be obtained in glycerine agar. The child improved, and was discharged from the hospital after about three months. At this time there was extensive atrophic paralysis of both upper arms. Re-examination eleven months later showed that the condition remained unchanged.

The author thinks that the case presents the clinical picture of an acute poliomyelitis, and not that of cerebro-spinal meningitis, since the characteristic symptoms of this disease were entirely absent, the only ones suggesting it being the somnolence, pain in the neck, and slight stiffness in the legs. He suggests that the infectious agent may distribute itself, on the one hand, in areas contiguous to the vessels in the meninges, and on the other, along the course of the anterior central artery of the cord. According to whether the meninges and nerve roots are chiefly affected, or whether the more central portions of the brain and cord are the main seats of the process, we get the picture of

cerebro-spinal meningitis or that of encephalitis or poliomyelitis, while mixed types may also occur. ALLEN.

127. EIN FALL VON TABES DORSALIS MIT BULBÄRPARALYSE (Tabes with Bulbar Paralysis). M. Bloch (Neurologisches Centralblatt, 18, 1899, p. 344.)

This condition, stated by Bloch to have been reported by two other observers only, Howard and Charcot, occurred in a man fifty-five years of age. Since 1890 he had the symptoms of a pronounced locomotor ataxia, beginning with lightning pains. Within the past year he noted symptoms of partial anesthesia and numbness in the right half of the face, and inside of the mouth and tongue. Swallowing was often accompanied by regurgitation through the nose, speech was nasal in character, chewing became difficult, and the saliva ran from the mouth continuously. There was partial ptosis of the right lid, paralysis of the abducens, superior and internal rectus, and paresis of the inferior rectus. The muscles of the left side were also partially paralyzed. Amaurosis and typical optic nerve atrophy were present. There was lessened sensibility over the right trigeminal distribution. In mouth and mucous membrane of the nose, right side, and the right side of tongue there was diminished sensibility. Smell, taste and hearing were intact. The tongue protruded with difficulty, bending toward the right side. The musculature was atrophied, and fibrillary twitchings were present. Paralysis of right posticus, with normal superior laryngeal. Sensibility of pharynx diminished. The rest of the clinical picture was that of a typical case of tabes. JELLIFFE.

128. UEBER LANDRY'SCHE PARALYSE (On Landry's Paralysis). Goebel (Münchener medicin. Wochenschrift, 1898, Nos. 30, 31 and 32).

The author recounts the various views with regard to acute ascending paralysis which have prevailed from the time of Landry up to the present day, and reports a case carefully studied by himself, of which the following is an abstract: A man of 30, who had had syphilis, four weeks after exposure to cold and wet, was suddenly seized with a flaccid paralysis of the legs. There was next involvement of both external recti of the eyes, then of the anus, without change in electrical reactions, paresthesia, sensory disturbance, or involvement of sphincters; with free sensorium, a febrile temperature range, and normal urine. The existing paralysis increased; both exterior and interior muscles of the eye became affected, as were also the face, jaw and tongue muscles. There was interference with swallowing and irregularity of the respiration; in the latter stages weakness of the sphincter vesicae, with slight diminution of sensibility towards the ends of the extremities, and death by asphyxia, fourteen days after the onset of the paraplegia.

The examination post-mortem revealed some fatty degeneration in the muscles of the extremities, in the diaphragm, and in some of the eye muscles, with a peculiar pigmentary deposit in the right rectus cruris. The Marchi method showed degeneration in some parts of the cauda equina, with disseminated changes in the medulla and pons, and a fine, dust-like deposit in the subcortical medullary layer of the central and occipital convolutions. The cord and peripheral nerves showed no degeneration.

The author regards the case as one of Landry's paralysis, with some unusual manifestations, and thinks that it shows, at any rate, that the lesions are not purely neuritic in all cases. In view of the

fact that he found the vessels of the cord rather overfilled with blood, he recommends the trial in such cases of some preparation of ergot.
ALLEN.

129. ZUR LEHRE DER SPINALEN NEURITISCHEN MUSKELATROPHIE (ATROPHIA MUSCULARIS PROGRESSIVA SPINALIS NEURITICA, BERNHARDT) (PROGRESSIVEN NEUROTISCHEN ODER NEURALEN MUSKELATROPHIE HOFMANN) (Spinal Neurotic Muscular Atrophy). E. Siemerling (Archiv. f. Pyschiatric, 31, 1898, 1899, p. 105).

Siemerling contributes a complete history, with report of microscopical findings, in a case of this interesting and rare form of muscular atrophy. The history is of a young man, 20 years of age, with no especial family hereditary influences. Up to the age of 5 years he developed normally. At this time there commenced an atrophy of the small muscles of the lower extremities, and two years later the hands commenced to be involved. The atrophy progressed until at the age of 13 he was unable to walk. He later developed hypochondriasis. At the time of examination his intelligence was fair; there was some immobility of the pupils to light, no changes in the fundus, and extreme atrophy of the muscles of all four extremities. Knee-jerks were absent. The right leg was in contracture at the knee-joint. No club foot. Complete paralysis in the lower extremities, less in the upper. Fibrillary twitchings of the intercostal muscles. Loss of electrical contractility for both galvanic and faradic currents. Psychically there was marked hypochondriacal depression, with delusions. The patient died in collapse.

The microscopical analysis showed degeneration of the posterior and lateral columns, especially in the dorsal and lumbar regions. Atrophy of the anterior horn cells, Clarke's columns and the anterior roots. The posterior roots were intact in their extramedullary portion, and in some parts of their intramedullary course. Simple degeneration of the sensory and mixed nerves, with atrophy of the musculature. The résumé of the microscopical findings was as follows: Disease of posterior columns, most marked in the lower dorsal regions. In the upper dorsal and cervical regions almost total degeneration of the central part of Goll's column. In the columns of Burdach the degeneration becomes markedly less as the upper cervical region is reached. There is degeneration of the lateral columns posteriorly and partly of the anterolateral columns, the lesions being most marked in the lower dorsal and lumbar regions and decreasing upwards. The cerebral ganglion cells showed no alterations. The muscles of the lower extremities were in a condition of extreme degeneration, the gastrocnemius showing advanced fatty changes. All of the peripheral nerves examined, brachial, radial, median, ulnar, sciatic, crural, peroneal, and saphenous nerves, were markedly degenerated.

The author further gives a critical digest of the various theories accounting for the disease, and, basing his opinion on this one case, believes that the disease is one of intramedullary origin, the posterior roots being affected after their entrance into the spinal cord. He does not exclude, however, a peripheral origin for the disease.

JELLIFFE.

- 130.—PARALYSIE OBSTETRICALE CONSECUTIVE A DES TRACIONS ASYNCLITIQUES SUR LA TÊTE (Obstetrical Paralysis from Manual Traction on the Head). Plauchu (Lyon médical, 88, 1898, p. 545).

Plexus paralysis due to difficult parturition generally occurs in breech presentations, or when severe traction is made with forceps. In the case reported the presentation was by the vertex and the head

was born spontaneously, but the shoulders were arrested. The nurse seized the head with her hands and with all her strength exerted traction upward—toward the right shoulder of the child. Immediately after birth paralysis of the left arm was noticed, the superior (first) cord of the plexus being the most severely injured. PATRICK.

131. CASUISTISCHE BEITRÄGE ZUR HIRNCHIRURGIE UND HIRNLOCALISATION, etc. (Clinical Contributions to Cerebral Surgery and Cerebral Localization). Ludwig Mann (*Monatsschrift für Psychiatric und Neurologie*, Vol. 4, No. 5, p. 369).

Mann reports a case in which two focal symptoms were observed after a cortical injury, viz., loss of sensation in the left thumb and index finger, and loss of power to sing. At the necropsy a cyst was found involving the second right frontal gyrus, and a small portion of the anterior central gyrus in the lower part of its middle third. It was not certain whether the cyst was due to the original injury or to operation. The loss of sensation in the thumb and index finger was explained by the involvement of the lower part of the arm center, but the absence of paresis in thumb and finger was striking. This is said to be the only case with necropsy in which amusia was not combined with aphasia. Mann believes that possibly a lesion of the second frontal gyrus may cause amusia. In his case the lesion was on the right side of the brain, and the patient was right-handed. SPILLER.

132. UN CAS DE SCLÉROSE EN PLAQUES A TREMBLEMENT UNILATÉRAL (A Case of Multiple Sclerosis with One-sided Tremor). P. Remlinger (*Rev. de Médic.*, Vol. 19, 1899, p. 244).

The case reported showed the following cardinal symptoms: There was an intention tremor of the upper and lower extremities, limited to the right side; exaggeration of the tendon reflexes, more marked on the right side; gait markedly spastic; tremor of the lower lips; diminution in sight, due to beginning optic nerve atrophy; slow speech, with monotonous and scanning cadence. These symptoms had been developing for the past six years, during which time the limitation of the tremor to the right side was remarkable, being so constant.

JELLIFFE.

133. UN CAS DE MENINGITE CEREBRO-SPINALE SIMULANT LE TETANOS (A Case of Cerebro-Spinal Meningitis Simulating Tetanus). *La Presse Médicale*, 24 Decembre, 1898 (Leroux & Viollet).

The patient, a man of 40, had noticed some stiffness in the lumbar region three days before admission, followed quickly by paralysis of the legs and difficulty in taking nourishment. When admitted to the hospital he was rigidly extended, the back being some distance above the surface of the bed. The neck was very stiff, and there was moderate trismus. From time to time he had paroxysms of pain, causing him to cry out. The temperature was 40° C., the pulse 120, and the respirations 40. The latter were almost exclusively diaphragmatic. The pupils were perfectly normal. A diagnosis of tetanus was made. Roux and Martin, of the Pasteur Institute, saw the case in consultation and pronounced it meningitis. The patient continued to grow worse and died on the second day. At the autopsy small collections of pus were found just back of the frontal convolutions, and there was intense congestion of the arachnoidal plexus to the plexus of the fourth ventricle. Cultures made by Martin showed the presence of the diplobacillus of Friedländer. The authors also report a case with very similar symptoms, the patient having the slight contraction of the extensors of the vertebral column, slight rigidity of the lower extrem-

ities, and only a slight degree of trismus. The latter symptom, however, increased, and was associated with dysphagia. Intracerebral injections of anti-tetanus serum were therefore made, and the patient recovered. LeRoux and Viollet lay great stress upon Kernig's sign, which consists in the impossibility of overcoming the flexion of the thigh upon the abdomen when the patient is seated. This symptom, according to them, never occurs in tetanus, and almost never fails in cerebro-spinal meningitis. SAILER.

134. HEREDITÄRE FORMEN ANGEBORENER SPASTISCHER GLIEDERSTARRE (Hereditary Forms of Congenital Spastic Rigidity of the Limbs). A. Good (Deutsche Zeitschrift für Nervenheilkunde, 13, 5 and 6, p. 375).

Good reports three cases (two sisters and one brother) of congenital, hereditary, spastic rigidity of the lower limbs, and of the upper also, to some extent, in two cases, with vesical symptoms. Sensation and the electrical irritability were normal, and muscular atrophy, excepting that from inactivity, was not observed. The tendon reflexes were much exaggerated. Mental symptoms were absent. Trauma at birth or premature birth was not the cause of the condition, and no indication of hereditary syphilis could be found. The symptoms did not increase after the sixth year. No necropsy was obtained.

SPILLER.

135. ATROPHIE DES CENTRES NERVEUX, DANS UN CAS D'ATROPHIE MUSCULAIRE ET OSSEUSE D'ORIGINE ARTICULAIRE (Atrophy of the Nervous Centers in a Case of Muscular and Osseous Atrophy of Articular Origin). C. Achard and Léopold-Lévi (Nouvelle Iconographie de la Salpêtrière, 4, 1898, p. 262).

Atrophy of the right lower limb was noted, following a chronic traumatic or tuberculous affection of the right knee, which began in the seventh year of life. The bones of the right lower extremity were smaller than those of the left. Death occurred from tuberculosis when the man was forty-one years old. The right side of the spinal cord and the right anterior horn were considerably smaller than the left in the lumbar and sacral regions, and the right anterior horn in these portions contained fewer ganglion cells. The spinal lesions were supposed to be due to the early age of the patient when the arthritis developed, and to the long duration of the process. Atrophy of the left paracentral lobe was noted. SPILLER.

136. L'ATTAQUE D'OPHTHALMOPLÉGIE MIGRAINEUSE (A Case of Ophthalmoplegia Occurring in the Course of Migraine). Leon d'Astros (La Presse médicale, 20 Janvier, 1898).

The patient, a woman, 69, who had previously been entirely free from headache or migraine, but had suffered from dyspepsia, some neurasthenic symptoms, and recent emotional disturbance, was suddenly attacked with severe headache. This disappeared in the course of a few weeks. A few days later, however, she was suddenly awakened in the morning with a violent pain in the head, located in the frontal region and the occiput. Upon arising she vomited. The next day it was noticed that the left eye remained closed, and this paralysis was still present when two days later the physician examined her. At this time the eye was completely paralyzed, the pupil dilated, and failed to respond to light or accommodation. The pains in the head were still very severe. There was no paralysis of the other nerves of the face. In the course of a week marked improvement had occurred, and a month later, when the patient was again seen, the eye was perfectly

normal. During the attack and for some weeks after, the patient suffered from severe thirst, and had almost constant polyuria. The urine was of low specific gravity, and contained a trace of albumin. d'Astros calls attention to the absence of any unilaterality of the pain, and suggests in explanation of the symptoms some disturbance of the arteries supplying the common motor-ocular nucleus. These arteries arise from the posterior cerebral, just below the front of the third nerve, and then pass into the cerebral peduncle to be distributed to the nucleus, forming an independent system. He assumes that the pathology of the condition is of the nature of an anemic infarct. SAILER.

137. L'ŒIL TABÉTIQUE (The Eye in Tabes). Gilles de la Tourrette (Soc. Med. des Hopitaux, 24 Feb. 1899).

The ocular manifestations of tabes are very numerous. They involve the retina, the external musculature, and even the lachrymal apparatus. Although so well known and so frequently described, the author thinks that he has discovered a new one. According to his experience, it is of sufficient value to have frequently enabled him, on mere inspection of a patient who presented no ataxic symptoms whatever, to make a diagnosis before going into the history. This sign consists in a very peculiar aspect of the eye, and is not to be confounded with simple myosis, which, when of punctiform character and situated on blue irides, always suggests tabes. In this class of patients the eye is brilliant, but expressionless; "the eye bright, but the gaze blank," is the author's way of describing the condition. This appearance is quite independent of the condition of the pupils, which is unvarying, and is especially easy of recognition in irides of a dark color. JELLIFFE.

138. SUR UN CAS D'HÉMATOMYÉLIE OBSERVÉ CHEZ UN NOUVEAU-NÉ (A Case of Hematomyelia in a New-born). D'Herbécourt (Journal de Médecine de Paris, 10, 1898, p. 314).

An infant delivered at term by forceps and with difficulty through a contracted pelvis was apparently dead, but by means of mouth-to-mouth respiration and tractions on the tongue was finally resuscitated. It was then discovered that there was no respiratory movement whatever on the right side of the chest. The child lived two and a half hours, and a careful dissection could discover no trace of traumatism affecting the structures of neck or thorax. Examination of the central nervous system revealed only hemorrhages in the cervical region of the cord, and at one point in this region a vessel showing endoperiarteritis. The hemorrhages made practically one continuous effusion about 6 mm. in vertical extent, and located at the base of the anterior horns.

The observation is of some interest in connection with similar findings by Schulze, and with the possible relation of hematomyelia to syringomyelia, asserted by this author and by Minor. PATRICK.

139. UEBER DIE FOLGEN DER SPINALEN KINDERLÄHMUNG AUF DIE HÖHER GELEGENEN NERVENCENTREN (Concerning the Effects of Infantile Spinal Paralysis on the upper Nerves Centers). M. Probst (Wiener klin. Wochenschrift, 30, 1898, p. 729).

The examination of the brains from persons whose limbs have been amputated has given most contradictory results in the attempt to locate the motor cortical centers by this means. The study of the spinal cord has been far more satisfactory. Gowers examined the brain of a man with congenital atrophy of the left hand, and found the middle of the right posterior central gyrus smaller than the corresponding gyrus on the left side. Microscopically, however, no anomaly could be seen. Edinger examined the nervous system of a man with congenital absence of the left hand and of a large part of the left forearm; the right

central gyri were small, but appeared normal when studied microscopically.

Probst reports a case in which infantile spinal paralysis occurred at the fourth year of life, and resulted in atrophy of the left upper and the right lower limb. The man died at the age of 68. The anatomical findings in the spinal cord were similar to those reported by others in this disease.

Only three cases of anterior poliomyelitis of childhood in which the condition of the brain has been studied have been reported (Sanders, Rumpf, Colella). In these three cases, as well as in Probst's, atrophy of certain parts of the brain was found. Sanders found atrophy of the central gyri and of the paracentral lobe of one side. Rumpf also found atrophy of the central gyri of one side, and in both these cases the atrophy was on the side of the brain opposite to the affected limbs. In Colella's patient the right leg was paralyzed, and both left central gyri, the left paracentral lobule, the uppermost part of the right anterior central gyrus, and the anterior part of the right paracentral lobule were atrophied. In Probst's case the central gyri of both sides, on the right side especially the upper part, were atrophied, and the left marginal and angular gyri were less distinct.

Sanders and Rumpf found no microscopical anomalies in the atrophied gyri, but Colella noted diminution in the number of nerve cells and fibers. In Probst's case the cortex was only half as thick as normal, the nerve cells were small, the white matter within the affected gyri was diminished in amount, and the neuroglia was somewhat proliferated. The internal capsule in the location of the pyramidal tract was small in the cases of Rumpf, Colella and Probst, and the fibers here were of small caliber. Probst, like the earlier writers, noticed that the pyramidal tract was atrophied throughout its course, and in his case both lateral motor tracts in the cord were small.

These findings seem to indicate that the central motor neurons do not develop normally when the peripheral neurons are destroyed early in childhood, but the existence of atrophy of the central neurons following injury of the peripheral in adult age remains to be demonstrated.

SPILLER.

140. A CASE OF PROTRACTED SLEEP EXTENDING OVER FIFTY DAYS.
Skerrit and Stewart (*Brit. Med. Jour.* No. 1970, p. 957).

A young man of 17 years, after severe study with insufficient sleep, complained for a period of six weeks of feeling tired on slight exertion, and then developed an overpowering drowsiness, which rapidly increased to profound and continuous somnolence. For ten days every effort was made to keep him awake, without success, and thereafter attention was directed, merely to the prevention of body waste. There was nothing suggestive of catalepsy, there was no anesthesia, and his general appearance was good. He lay in a calm, placid sleep uniformly, and when roused to take liquid nourishment (every four hours) he would speak, in reply to direct questions, very much as one does who talks in his sleep. It would sometimes take ten minutes or a quarter of an hour to rouse him sufficiently to prevent his being choked in the attempt to get down the egg and milk, or beef-tea, or other nourishment. Indeed, he would frequently drop off to sleep while in the act of putting the cup between his lips. The sleep was never a "profound" one, and when the calls of Nature demanded his attention there was an obvious desire to respond to them; but he would frequently drop off to sleep again before he could be assisted and the inclination to evacuate his bowels or bladder would then disappear. Gradually this partial response ceased and he passed in bed his mo-

tions, which cascara sagrada kept fairly free. Every effort was made to get him to empty his bladder naturally by propping him up against the wall and otherwise, but it was generally a failure. During this period priapism was very marked, but there were never any seminal emissions, and his hands had to be tied to the bedstead to prevent his practising onanism—a habit to which he had never been addicted. He remained in this condition till two months after the onset; then he began, while half awake and half asleep, to talk as if in a dream, sometimes saying things which were ridiculous, and occasionally asking how long he had been ill. He also showed evidence of an emotional tendency unusual to him. His weight, which previously had been gradually decreasing, began now slowly to return to the normal condition, and he was allowed to sit up for an hour or two daily. The sense of taste, which was apparently quite absent previously, also gradually returned, but it was not entirely restored for some time.

For several months after the mental faculties were completely restored the patient had considerable difficulty in walking, on account of incoördination and a tendency to fall to one side. Recovery was ultimately complete.

The author says that only eighteen other cases of such protracted sleep are on record, and of these four died in the sleep. Weir Mitchell has given an epitome of these eighteen cases in the *Transactions of the College of Physicians of Philadelphia*, Vol. III, and more information on the subject will be found in Gould and Pyle's "Anomalies and Curiosities of Medicine."

PATRICK.

THERAPY.

141. VERSUCHE ÜBER COCAINISIRUNG DES RÜCKENMARKES (Cocainization of the Spinal Cord). Bier (*Deutsche Zeitschr. für Chirurgie*, April, 1899).

The author reports the results of some remarkable experiments that he has been making to test the feasibility of rendering large areas of the body anesthetic by cocainization of the spinal cord. The method employed was to throw minute quantities of the drug into the subdural space after the manner of Quincke's lumbar puncture. By the use in this way of 0.005-0.01 gm. of cocaine he was enabled to perform painlessly such operations as resection of knee and ankle-joints, necrotomy of the tibia, resection of the ischium, resection in a case of complicated fracture of the femur, and treatment of an osteomyelitis femoris. As drawbacks to the procedure there were manifested in several cases disquieting symptoms, such as severe headache, nausea and vomiting, which lasted for several days. In order to estimate the intensity of these more exactly the author tried the injection both on himself and his colleague Hildebrand, finding that anesthesia of the whole of the lower extremities set in after an interval of from 5 to 8 minutes. A dose of 0.005 gm. maintained this state for about 45 minutes, then sensibility slowly returned. The after-effects were marked on both experimenters. Bier was even obliged to remain in bed for several days. Before making further trials on the human subject the author intends to investigate more fully the effects on animals.

JELLIFFE.

142. HYDATID OF THE BRAIN—OPERATION—RECOVERY (Vance, *Australasian Medical Gazette*, Feb. 21, 1898).

A man of thirty-six was admitted to hospital with right hemiparesis and aphasia, the trouble having begun six months previously with twitchings of the tongue and arm, gradually followed by the other

symptoms. For the first few months of his illness he suffered from epileptic fits, exact character not stated. There was no loss of sensation, no headache, and no optic neuritis. Under medical treatment there was no improvement and no aggravation of the symptoms, except that six months after admission he was found to have double-choked disk. Mr. O'Hara operated, finding a cyst about the size of a bantam's egg, which was drained. Patient made a good recovery from the operation. In discussing the paper Mr. O'Hara reported another case of hydatid of the brain in a boy operated upon eight years before. He had made a perfect recovery from a complete left hemiplegia and double optic neuritis.

PATRICK.

143. L'ÉLONGATION DU PNEUMOGASTRIQUE (Stretching of the Pneumogastric) Jaboulay, (*Lyon Médical*, Vol. 87, 1898, p. 537).

The author, who has been almost fantastically active in surgery of the nervous system, now reports upon stretching of the pneumogastric. He says that this measure may modify the excitability of the bulbo-pontine region by traction upon the fibers of origin, and also that of the branches of the nerve distributed to the respiratory, digestive and circulatory mechanisms. He has performed the operation for one case of epilepsy, proceeding upon the assumption that Schroeder Van der Kolk was correct in localizing the convulsive center in the locus ceruleus, and believing that the operation would "modify" this region. The patient was a boy of sixteen years, who had as many as twenty-five attacks a day, preceded by an epigastric aura, and who was relieved by the operation, it is not said for how long nor to what extent. The author claims the peculiar advantage of this operation over others that it is particularly adapted to the large number of cases of idiopathic epilepsy that "begin in the epigastric region." He further asserts that when this operation on one side is combined with unilateral section of the cervical sympathetic that the peripheral cardiac effects are compensatory and the encephalon is modified in its entirety. He has also stretched the pneumogastric in a case of exophthalmic goitre in which section of the sympathetic had been previously performed. Although the first operation relieved the exophthalmos, it had no effect upon attacks of coughing which greatly troubled the patient. These were relieved by the pneumogastric operation.

PATRICK.

144. UEBER SUBCUTANE INJECTIONEN VON HEROINUM MURIATICUM. (Heroin used subcutaneously.) A. Eulenberg (*Deutsche med. Wochenschrift*, March 25, 1899. p. 187).

The muriate of heroin, a new derivate from morphine, has been used extensively by this well-known clinician in some three hundred cases. It is readily soluble in water, and is used in two-per-cent. solution. The maximum dose should not be over 1-6 grain for an adult. Therapeutically it is indicated as an antispasmodic in asthma, and is of service in various neuralgias, intercostal, trigeminal, and sciatica, the myalgias and arthralgias. It has proven especially valuable in cases where chronic morphine medication has lost its value. His experience would also seem to bear out that of other recent observers, that heroin is destined to become a very valuable drug in the treatment of chronic morphinism. The daily dose should not exceed $\frac{1}{2}$ grain.

JELLIFFE.

Book Reviews.

DISEASES OF THE EYE. A HANDBOOK OF OPHTHALMIC PRACTICE FOR STUDENTS AND PRACTITIONERS. By G. E. de Schweinitz, A.M., M.D., Professor of Ophthalmology in the Jefferson Medical College, etc. Philadelphia. W. B. Saunders, 1899, pp. 700, with 255 illustrations and 2 colored plates.

This new edition of de Schweinitz's text-book is the third since its first appearance in 1892. It contains much new matter and is brought well up to date, forming a representative exposition of the art of ophthalmology as practised in this country to-day. Without going deeply into questions of pathology, or entering upon prolix discussions of unimportant matters, it offers concise, clean-cut descriptions of disease, and detailed, practical rules of treatment. There is noticeable an absence of repetition, and also of that haphazard jumbling together of undigested extracts from different sources which too often characterizes the larger text-books, when the author, aiming at completeness, usually arrives only at confusion. The chapters of chief neurological interest, those on affections of the optic nerves and on disturbances of the ocular muscles, while not long, are clear and to the point. The matter in the book is arranged systematically and in a way that is convenient for reference. Descriptive terms have been well chosen, as a rule, and the purist in language will only regret that the author has followed a common German practice in employing the hybrid word *pericorneal*, when he had at his service either the pure Latin derivative *circumcorneal*, used by English writers, or the pure Greek derivative *perikeratic*, used by the French.

WARD A. HOLDEN.

DIE LEITUNGSBAHNEN IM GEHIRN UND RÜCKENMARK. By W. v. Bechterew. Second edition, completely revised and enlarged. German, by Richard Weinberg. Leipzig: Arthur Georgi, 1899.

v. Bechterew's book is an old friend in a new guise. The volume with which we were all familiar has grown to two or three times its former size; new material has been added, and the old has been rewritten. v. Bechterew is one of the most voluminous writers in the world, and he excites our wonder by the amount of original work he is able to accomplish. His "*Leitungsbahnen*" represents his own investigations and those of the students in his laboratory, but neurological literature is by no means ignored. It is not a mere compilation from other books, but bears the unmistakable stamp of original thought. It is a work on anatomy that one may be glad to consult, even though he may hold different views from the author.

The chapter on the conducting tracts of the spinal cord differs very considerably from the corresponding chapter in the former edition. Many disputed points are mentioned. Fibers from the column of Clarke and from the central cell groups of the gray substance are said to pass through the anterior commissure to the opposite side of the cord, although some fibers from these cells go to the white matter of the same side. All these fibers are closely connected with the posterior roots, and the theory of a partial decussation of the sensory tracts within the spinal cord by means of a second series of neurons seems to have some support. This would explain better than anything else the Brown-Séquard symptom-complex.

v. Bechterew refers to some experiments done in his laboratory, in which ascending degeneration was found in the lateral ground bundle of each side of the cord after the posterior roots in the dog

were divided. This was evidently a degeneration of central sensory neurons following a lesion of peripheral, and as such is remarkable. It seems strange that a similar condition has not been observed in man. In experimentation there is always the possibility that the anterior portions of the cord may be injured in attempting to cut posterior roots. We may be pardoned for desiring further confirmation of the statement that central sensory neurons degenerate quite rapidly, or even slowly, as a result of lesion of peripheral sensory neurons. This whole subject is at present under discussion, and Van Gehuchten has found it necessary to modify certain statements made by him which seemed to indicate that such secondary degeneration in sensory neurons occurs.

It is important to bear in mind in doing experimental work that in higher vertebrates, even the dog and the cat, an anterior pyramidal tract does not exist, and that in the place of this an uncrossed lateral pyramidal tract is found. The German reads almost as though the statement were made that the pyramidal tract in those animals does not decussate: . . . "fehlt eine vordere Pyramidenbahn vollständig, statt dessen ist ein ungekreuzter Pyramidenseitenstrang vorhanden." v. Bechterew evidently does not intend to deny the decussation of the motor fibers.

The presence of the "intermediate bundle" within the crossed pyramidal tract, and the origin of this bundle in the cerebellum, seem to lend some anatomical support to the theory that motor fibers (Van Gehuchten) pass from the brain to the spinal cord by way of the cerebellum. v. Bechterew believes that in addition to the "intermediate bundle," a tract exists, anterior to the crossed pyramidal tract, having its origin in the thalamus and descending to the cord through the Forel decussation.

The fibers described by v. Bechterew as the olivary tract seem to have their origin in the lower olive, although this has not been positively determined.

According to v. Bechterew, the "pupillary fibers" pass with the optic nerve to the chiasm, decussate partially here, ascend in the optic tract to the external geniculate body, and reach the oculomotor centers, with partial decussation, through the posterior portion of the thalamus and the posterior cerebral commissure. An isolated lesion of the external geniculate body, or of the anterior corpora quadrigemina does not destroy the reaction of the pupil to light.

v. Bechterew believes that different fibers exist for the different forms of sensation; the fibers of the lateral columns conducting sensations of pain and touch, and those of the posterior columns the muscular sensation.

He mentions that attempts have been made to determine the motor areas in the human cortex by electrical irritation, but he does not allude to the fact that American investigators have been especially active in this form of experimentation. The carefully written paper by Lemacq gives proper credit to our countrymen for information obtained by this method.

It is a strange fact that the motor fibers for different portions of the body are separated within the internal capsule, but that in the lower parts of the central nervous system they are intimately mingled. This, however, seems to be the generally accepted teaching.

The lateral bundle of the cerebral peduncle is said to arise in the temporal lobe and, to a less extent, in the occipital lobe; in this v. Bechterew differs from Dejerine.

Space does not permit a thorough review of all the interesting statements found in this book. It is a volume of 692 pages, is profusely and well illustrated, and contains very many references to the literature. Every part of the central nervous system receives a very careful study.

SPILLER.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

AMERICAN NEUROLOGICAL ASSOCIATION.

Twenty-fifth Annual Meeting Held at the Hotel Dennis, Atlantic City,
N. J., June 14 and 15, 1899.

The President, Dr. James Hendrie Lloyd, in the chair.

PRESIDENT'S ADDRESS.

Gentlemen:—

In assuming the duties of the distinguished position to which you have elected me, I can and ought to do no less than express my full appreciation of the fact that I am thus indebted to you for one of the highest honors that can be conferred in his own specialty and his own country upon an American neurologist. This debt is not lightly assumed by me, the more so that I am conscious that it cannot be fully discharged. My conception of the dignity and the position of this association in the field of neurology, especially in this country, is so high that I trust I may be pardoned if, with some feeling of self-consciousness, I express to you a keen sense of responsibility at the same time that I acknowledge to you my indebtedness.

The twenty-fifth anniversary of the association naturally suggests a reminiscent mood. I shall not deal in reminiscence, however, because that task has been allotted to another. I shall stay in the present and look to the future. I think that this annual meeting should always be the occasion for the association to carefully consider its own progress and aims, and that he who is called to preside should be the mouthpiece for some carefully considered utterances touching, as it were,

the personality and welfare of our organization. It is said that Epicurus of old dispensed his sages from all foresight and care for the future, but this is not in accord with the philosophy of the nineteenth century. We have to look onward as well as to look about us, and the better injunction is that of Plato—"Do thine own work, and know thyself."

In the field of neurology in America to-day it is essential that there should exist such an association as this to act as an exponent of one of the most important of the specialties. This society represents a centralizing tendency, which is rather in opposition to the localizing spirit of the Anglo-Saxon. In science, as in politics, the danger in this country is rather in a spirit of local self-government carried to extremes. Our risk, although it may not be a great one, is rather towards provincialism—and provincialism never yet accomplished great things. If too much of this is bad in politics, it is infinitely worse in science; and our country, with its vast extent and multitudinous interests, presents a territory in which the truly national association is needed as a court of Sanhedrim or court of final expression. To the somewhat wabbling and loose-jointed machinery of some of our schools and societies—in which neurology goes as it pleases—this association may act as a sort of balance wheel, whose function should be most salutary. If I were to seek for the most honorable appellation for this body, I should like to say that it is truly *national*—not national in the petty and narrow sense that it is evenly represented in the cities and States, but national in the sense that it includes every neurologist in this country of approved (and only approved) attainment. There is a difference between an association being geographical and being national. We do not regard the geography of the States. Personal attainment, not residence, is alone the criterion. If these thoughts are trite, they are none the less vital, because a strict regard for them can alone secure the success and vitality of this society. Let them be repeated from this chair every year, just as the ancient king bade his servant remind him every day that he was mortal.

In most of the genuine original work in neurology done in the United States for the past 25 years, this association is represented by its members. In other words the amount of en-

during neurological literature that has been contributed by men whose names are not, or never have been, on its list of membership is surprisingly and conspicuously small. No national association of specialists in America can, by this standard, claim to be more truly representative. And that this standard is the correct one is evident from the fact that neurology is in a special way a most difficult and involved field of practice, and that the man who reaps genuine success in it is, as a rule, only too willing and anxious to be admitted to associate with those who are already identified with it. In other words, he feels that in this association alone can he meet with both the understanding and criticism which are at once the reward and the stimulant of his own work. The man who works in nervous diseases and never seeks to gain membership in this association, must either have some secret misgivings about his own neurology, or entertain an indifference toward the very specialty which he professes. Here alone is the arena in which he meets his true confrères; here alone can he hope for the spirit which makes a symposium of his nocturnal vigils in neurology, and creates an atmosphere which is productive of his best thoughts. Whatever our regard for other societies may be, we must all recognize the fact that profound neurological lucubrations are often still-born when they are produced in other associations than this, and the character of the discussion which greets them, in such alien surroundings, must often convince us all that it is as important occasionally to recover one lost sheep for its own good as it is to sit down and rejoice over the ninety-and-nine who never wander away.

The stimulus which this association can give to the creation of a high standard of neurological work in this country is, I conceive, well nigh incalculable. One of its most important functions, and one which has not always been recognized at its full value, is to stimulate research and to elevate the prestige of the American name. It should be the prerogative of this society to inspire ideals, on the one hand, and to pass final verdicts, on the other. There is still some tendency to do crude work in America, probably because of the very exuberance of our conditions. With a practical genius unexcelled by any

other people, we have not excelled in all the mechanical arts of scientific medicine. It is still too much our custom and our need to look abroad for some of our technique. Who can doubt, who knows thoroughly the genius of our people, that this will not always be so? Who can hesitate to believe that the same genius that created the telegraph and has brought to perfection the devices of electricity can also attain to the highest accomplishments with the microscope? Cannot the mind that grasps the most intricate problems of steam, of electricity and of mechanics also contribute successfully to the most involved questions of bacteriology and pathology? If it be true that we are standing on the threshold of a new era of national achievement and expansion with the dawn of the new century (as some men claim who still feel the impulses of the late war in their veins) then let us hope that our own science is about to partake of renewed energy along yet novel lines, and that this association will be the recognized theater for some of its best display.

While this association is so potent in the neurological literature of this country, it is to be somewhat regretted that its influence is not yet paramount in neurological education. It is true that many of its distinguished members are in positions of clinical importance in some of our leading schools, and are thus contributing to the proper neurological training of a future generation of physicians. They represent, as it were, a compromise between the new and the old, and the schools which they serve are deservedly in the front rank of progress. Unfortunately this is not universally so, and we still witness the incongruity of didactic teaching in neurology being done by general clinicians who make no claim, outside of their professorial chairs, to being expert neurologists. The methods that prevailed one, and even two, generations ago, before nervous diseases were recognized as an intricate specialty, are still permitted to prevail. I trust I do not transgress the limits of fair and accurate criticism when I say that the professors of general practice in all our medical colleges for the last twenty-five years have, with one or two noteworthy exceptions, contributed but little if anything to the store of neurological literature. However distinguished they may be in general internal pathology,

they seem as by common consent to avoid the neurological fields as though these were sown with noxious weeds. In their own schools there is usually a clinical professor who does the actual practical work in the nervous clinic, while they confine themselves to the erudite pages of venerable written lectures. The cause for this is not far to seek. Neurology is a jealous mistress and will not brook numerous rivals. The old-fashioned professor of practice is simply not proficient in neurology. He has not kept abreast of its progress, and does not seem to care to do so. He is (with few exceptions) not familiar with its clinical details, its involved morphology and its expensive and intricate technique. He is usually frank enough to admit this in the consultation room and on the street, but when he retreats into the sacred arcanum of his lecture-room his rôle is instantly changed, and he assumes all the dignity and claims all the privileges of the truly initiated. Why he should be willing to attempt to teach neurology by his didactic methods, and why others should be willing to leave him do so, are mysteries that can only be explained by the force of custom and the tyranny of prejudice. These things should not be so. There is no monopoly in science, however there may be monopolies in medical politics; and we have the right in this association to claim at least that the eminent men who essay to teach neuropathology from the chairs of general practice should sometimes prove both their ability and their zeal by the amount and value of their original work in this field. We would all welcome their contributions not only for such intrinsic merit as they might have, but also as an evidence that the department of nervous diseases was not being slighted for other departments to the detriment of both the profession and the students.

I think it will be granted by the majority, that while the didactic lecture still has and probably always will have its proper place in the curriculum, it is no longer fit to be the main-spring of instruction in nervous diseases. Instead of having first, it should have second place. It is inconceivable any longer that medical students can learn neurology by listening to a few time-worn lectures. The bedside and the laboratory have usurped the place of the lecture room; and the school that does not recognize this fact is not just to its pupils. The

schools, to be sure, are recognizing it more and more, but in some places there is evidently much yet to be done. From considerable observation among young medical graduates I have for a long time seen clearly (just as they themselves discover when they enter the hospitals as internes) that they are less well equipped in neuro-pathology than in most other branches. This is no doubt partly due to the difficulty of the subject and the fact that it has not been made interesting in the medical schools; but primarily, I believe, to the fact that too much importance is still given to the didactic lecture, and that this is still included in the course of general medicine.

It was the first aphorism of Hippocrates that "Experience is fallacious and judgment difficult." Certainly, it is not my ambition in this place to attempt to cast a horoscope for the science which we all profess.

The science of neurology presents several phases at this time which are worthy of special note.

In the first place, if I mistake not, it has grown somewhat lukewarm with reference to the revelations of bacteriology as affecting problems arising in its own domain. The reason for this may be due in part to some disappointment in not having realized as much as it expected from the doctrines of infection. Certainly, from a therapeutic standpoint the returns have been coming in rather slowly from the bacteriologists, and we are not prepared yet to say that we have a reliable serum treatment for many nervous diseases. We cannot to-day re-echo the optimistic sentiment of a former occupant of this chair, that we would yet put a stop to the funereal march of our patients through our consulting rooms. If the hypodermic syringe, loaded with animal serums of every grade and variety, is to do all this, let us bid good speed to the day when neurology will be but the handmaiden of her younger sister, bacteriology, and the syringe will have usurped the place of the microscope in our affections. In pathology, however, there is no occasion for disappointment, for it seems almost certain that we are at least on the threshold of a wide new field in cytology, in which much that will be done will probably owe much to our advanced knowledge of infection and of vito-chemistry.

While fully in sympathy with much of the enthusiasm which

at present marks the study of the nerve cell, I think I can see in this enthusiasm some of the extremes which periodically mark the progress of the medical sciences. We move in cycles and epicycles in medicine, and like some primitive tribes we always worship the new moon. We are just now engaged in the cult of Nissl; we are at the high tide, as it were, of methylene blue. At another time it was the neuron-theory which threatened to redeem our science. Again, we are called to trace the invisible fibrils of Apáthy, which we are assured, will yet bind together not only a disjointed nervous system, but also a fragmentary science. I make no doubt that some of us are too much given to extremes in some of these matters, and may see more promise through the small end of a microscope than Moses saw from Pisgah. It is not exactly a good thing to use the lamp of Aladdin to illuminate the field of a microscope. We should never forget that in our histological researches we are simply numbered among those who from time immemorial have been seeking to read in dead matter the mysteries of life. The problem which has forever refused to give an answer to the Hindu and the Greek, to a Newton and a Leitnitz, will probably not yield to our more importunate appeals, and the dead will continue to refuse to give up its secret of life. The fact that we have a new stain that will depict the contours of chromophilic bodies, does not alter the fact that those chromophilic bodies are still without assured physiological value, and that when we look upon them we may be merely looking upon the products of our own clumsy handiwork. We may think that we are detecting life in its innermost recesses, whereas we are merely gazing upon the wreckage of the temple.

This is not merely the pessimism of a despairing medical philosophy. Such considerations, if rightly apprehended, may serve to remind us of our true functions as investigators of disease. Pathology, it may be rightly contended, is not the science of life, but of disease; and we accomplish our true aims if we, like the archeologists, can point out unerringly what has been the normal structure from a study of its remains, and can do something to reconstruct the ruins, even if we are not able to grasp all the mysteries of the original creative force.

In the domain of the nervous system this is surely a fascinating study, for in that tissue is manifested the highest functions of organic matter.

I should think it amiss if I did not say something here about what seem to me to be some of the characteristics of contemporary neurological work in America. In the first place, there is apparent a wholesome tendency to greater self-reliance and originality in our work. He would be a blind and prejudiced critic who hoped for or advocated a distinctive national science of neurology. Such a claim or hope would be the worst kind of a solecism—the most humiliating form of national vanity. Science, in all its fields, is truly international; and has, indeed, come to be one of the mightiest bonds which unite the nations. It proclaims the unity of the race, and does much to break down the barriers of mere political, ethnic and religious differences. We can, however, recognize the fact that each nation may elaborate its own methods of work, and out of its own distinctive genius evolve its own indigenous schools, which best prove their vitality and their right to exist by the value of the contributions which they make to the common store. The science, like the literature, of a people must, in a sense, be its own, if it is to be worthy of acceptance by other peoples and by posterity. We cannot remain mere copyists and disciples of foreign masters, if we are to vindicate our own originality and our own status. A vigorous competition of the nations in the domain of science insures a fulfilment of the universal law of the survival of the fittest, and thus contributes to the evolution of enduring forms. In this vital contest it is surely our spontaneous hope that America will yet be among the foremost in neurology. She can only be so by recognizing and yielding herself to the inexorable law that the survivor in this active struggle is the one who adjusts himself to his own and not to a foreign environment.

Must a man inquire anxiously when he undertakes to do original work whether he is in exact line with the French or has oriented himself accurately to a German standard? I think it has been from a rather nervous impulse of this kind that there has existed, especially in the past, a tendency for some of our writers to overload their pages with bibliographical references.

It is notorious that the French and Germans themselves (although the latter especially are careful bibliographers) do not load down their pages in this way as do some of our more conscientious and younger writers. The futility of such carpenter-shop methods (of which we have all been guilty) is now glaringly apparent, when one takes but a glance into the "Index Catalogue" or the "Index Medicus." Any foot-note bibliography pales its ineffectual fires before these mighty tomes, and proclaims its own inadequacy. With the daily increase of medical literature, no man can be thorough, if by that is meant that he must exhaust everything that has gone before him. Moreover no man's work can any longer be altogether original; he must do over again what others have done in part before him, and his own original contribution may be but as the grain of wheat in the sheaf. The great astronomer, Laplace, recognized this fact so fully that it is said he appropriated from everybody right and left, and gave no man credit for anything. Thus is he accounted a genius. Shakespeare stole his plays (so it is said) and many a poet has been but a microbe in the body of a dead Homer. Why should a man laboriously overload his literary work with references which, if they prove anything, prove that he is a copyist? Foot-note references are like tombstones; they are simply monuments of other men's remains; the older they are the more they are apt to be out of place, and their record is often false. Montaigne said that if you took away his quotations you took away himself—and so it is with some of our scientists. I believe some reform in literary method in favor of brevity, perspicacity and the aim to say what a man sees and knows for himself (and not what he thinks some other men have seen and known) is what we have some need of in our American neurology.

Finally, let us not forget that the great lights in medicine have been the great clinicians. If we need a special reminder of anything in our American neurology it is that we should not despise being clinicians and therapists. A neurologist should be willing to do something for his patients before they are dead. Our programme this year shows a striking dearth as usual of therapeutic interest. The fine scorn of the professed microscopist for the giver of drugs is shown here in

unmistakable titles. We are all immersed in what we choose to call science, and have not much eye for art. Has the theory of the neuron helped us to make a single diagnosis? Will the claim of Apáthy either relieve or cure? While I would not ask any man to belittle science, I trust we will not allow science to belittle us. If we do not constantly keep expert in reading the signs of disease at the bedside, and ministering to the wants of the suffering, our medicine will surely become a dry and barren thing.

145. ON THE INCREASE OF INSANITY. T. S. Clouston (Eighty-Sixth Annual Report of the Royal Edinburgh Asylum, 1899).

Clouston, in this last report of the asylum, among other things comments upon the question of the so-called increase in insanity. "It seems to him that the number of admissions to hospitals for the insane will increase for many years to come, *not* from any increase of insanity at all, but from a more extended realization among society, of every grade, of the benefit and convenience of such hospitals. It is getting better understood that many forms of mental disease are just morbid accentuations of natural disposition; in one case temper shading off into mania; in another, keen sensitiveness of feeling passing into melancholia; and in a third, suspiciousness verging into insane delusions; so that the subjects of such changes become unfit for family or social life." "The world is getting too busy to be able to attend to its mental breakdowns at home, and it is getting more intolerant of very marked divergencies from social order, and even neglect of the conventionalities of life."

JELLIFFE.

146. TABETISCHE KRISEN MIT HOHEM FIEBER (Tabetic Crises with High Fever). P. K. Pel (Berliner klinische Wochenschrift, No. 26, 1899, p. 561).

Pel describes a new sign of tabes under the name of "crises fébriles" (fébrile crises). A tabetic patient had several attacks of severe pain in the extremities, with high fever and vomiting, free perspiration, etc. One attack observed in the clinic is described in detail. The man awoke after a good sleep, feeling a little unwell. About half past eleven in the morning he began to feel chilly, without having a distinct chill, and to be very sick. Attacks of severe pain then began in the feet and extended to the lower part of the legs, and later to the upper extremities. Painful twitchings developed; the legs were drawn up and the hands were stiff. Pain was also felt in the trunk. Repeated vomiting occurred in the afternoon, when the pain was most severe. Toward evening severe pain in the eyes, intense photophobia, secretion of tears and congestion of the head were experienced. Both eyes appeared red and inflamed, as though they had been rubbed severely. By the following morning the ocular symptoms had disappeared, the pain was more endurable, and vanished within the next twenty-four hours. The temperature during the attack reached 40.2 degrees, and the pulse 150 beats per minute. Herpes labialis was observed on the day following the fever. No enlargement of the spleen was noticed after the attack. The man had had five of these seizures, all very similar to one another.

SPILLER.

EDEMA OF THE PARALYZED LIMBS IN HEMIPLEGIA, WITH REPORT OF AN UNUSUAL CASE.

BY CHARLES LEWIS ALLEN, M.D.

CLINICAL PROFESSOR OF NERVOUS DISEASES IN THE GEORGETOWN UNIVERSITY, WASHINGTON, D. C.

In hemiplegia from gross brain lesion it is not uncommon to find some swelling, with glossiness and slight pigmentary changes in the skin of the paralyzed limbs; less frequently muscular atrophy and changes in the joints occur, but edema to any such extent as was observed in the following case is excessively infrequent:

Matthew K., 55 years old, black, family history and previous personal history unattainable, is said to have had a slight apoplectic stroke several years ago, but to have recovered with but little disability. On December 15, 1897, he was noticed to be acting queerly and to have difficulty in finding his way about, so he was kept in the house. Two days later, as he attempted to arise from his chair his son noticed that his right arm and leg were paralyzed, but thinks he never lost consciousness.

Power was never regained in the paralyzed limbs, and the swelling, to be later described, came on gradually several weeks after the onset of his attack. He entered the Freedman's Hospital on March 17, 1898. It was then noted that he had a complete left hemiplegia, with swelling of the affected limbs, but no contractures.

He was first seen by the writer on October 12, 1898, when he lay helpless in bed suffering from marasmus and chronic diarrhea.

Examination October, 1898, showed a large, quite emaciated black man, completely bedridden. His mental condition was dull, but he could be gotten to reply to questions, and complained of pain in the paralyzed arm. The two last phalanges of the first finger of the left hand had been lost by an accident in early life. There was complete paralysis of the left arm and left leg. On movement the face was drawn somewhat to the right, the tongue deviated to the left. The head was drawn some distance toward the left side, and fixed in this position by a contracture. The left arm was partly flexed at the elbow, and pressed so firmly against the left side that an

ulcer had formed over the ilium. The fingers were pressed into the palm. The left leg was partially flexed at the knee and everted. The limbs were held in these positions by contractures. The left arm and hand were greatly swollen. The swelling extended from about the junction of the upper and middle thirds of the upper arm, being most marked in the forearm and hand. The whole arm felt boggy and pitted on pressure, but the swelling was painless and there was no inflammation. The bones were apparently unaffected. The swelling fell off rather sharply at the point indicated (junction of the upper and middle thirds of the arm). The skin over the



affected region was harsh and glossy, and on the posterior surface of the arm and hand there were areas of increased pigmentation, some as large as a silver dollar. The left leg and foot were also edematous and swollen. The swelling extended above the middle of the thigh, but was not so marked as in the arm and hand, nor were there any pigmentary changes. There was no edema of the face. The left side of the trunk appeared, perhaps, a little fuller than the right, but on account of the condition of the patient no satisfactory measurements could be taken. There was no edema of the scrotum. There was some wasting of the right arm and leg, but no other changes on that

This photograph is not altogether satisfactory from an artistic standpoint, but it has been reproduced because it shows very well the degree and extent of the edema.—ED.

side. The following measurements were taken at corresponding points on the two sides:

Right arm.	Left arm.
Below shoulder22.5 cm.27.5 cm.
Middle of upper arm 21.7 "32.5 "
At elbow23.7 "35.7 "
Middle of lower arm 19.3 "28. "
Wrist14.8 "18. "
Around hand21.8 "25. "
Length of hand21.8 "21.8 "
Right leg.	Left leg.
Around great trochanter47.2 cm.49. cm.
Middle of thigh35.7 "39.7 "
Knee36.3 "40.2 "
Middle of leg27.5 "33.1 "
Ankle22.5 "25. "
Around dorsum of foot26.1 "28. "

There was no eye muscle paralysis. The pupils were even, but did not react for light or accommodation. Speech was unaffected. On account of the contractures the reflexes could not be obtained, and because of the mental condition of the patient no satisfactory examination as to sensory disturbances could be made. The heart sounds were rather weak, but there was no murmur, and no alteration in the area of cardiac dullness could be detected. The arteries were somewhat rigid and thickened. Careful examination gave no evidence of any thrombosis in the vessels of either arm or leg, nor was there any phlebitis. A few bronchial râles were heard over the chest. Examination of the abdomen was negative. No urine could be obtained. The accompanying photograph was made October 21, 1898.

The patient's diarrhea continued; he grew progressively weaker, and died on October 24, 1898.

Autopsy, October, 25, 1898, seventeen hours after death, by Dr. D. S. Lamb:

Body of large black man, slight bed sore over left ilium; nutrition poor; some wasting; left arm and leg swollen and boggy, but swelling seemed somewhat less than before death. On opening the chest the right lung was found edematous, the left lung collapsed and compressed against the spinal column. The left pleura showed old adhesions, and some recent pleurisy;

with exudation of plastic lymph; and contained about a half pint of serum, slightly blood-stained. There were some recent pericardial adhesions, and inflammatory lymph exudate, especially marked on the left side. Dissection over the region of the axillary, brachial, and femoral vessels showed neither phlebitis, thrombosis, nor any growth which compressed the vessels, nor could any tumor be found in either the thoracic or abdominal cavity. The liver was congested, the spleen small and pale. The kidneys were enlarged, rather congested, and seemed to have some increase in their connective tissue. The stomach and intestines were distended with flatus. There were some spots of congestion in the colon, but no ulceration.

Nervous System.—There was great difficulty in getting permission to open the skull, and removal of the spinal cord was not allowed.

The calvarium was of normal thickness, the dura somewhat adherent to the skull along the superior longitudinal sinus. The pia-arachnoid showed some milkiess along the vessels. The whole right hemisphere felt softer than the left, especially in the Rolandic region, about the ascending limb of the fissure of Sylvius, and in the island of Reil. There were marked areas of softening about the lower end of the fissure of Rolando. In the posterior portion of the parietal lobe and in the occipital lobe there were areas of softening, yellow in color, and about as large as a silver dollar. On horizontal section a large cyst was found on the right side, having well-defined walls and filled with reddish-colored, grumous material. This cyst occupied all of the internal capsule, had destroyed most of the lenticular nucleus, and passed forward into the frontal and backward into the occipital lobe. The arteries at the base appeared to be of good caliber, and there was no other trouble found there. The cerebellum was normal.

Microscopical Examination.—Pieces of the cortex in the Rolandic region of both sides, the whole brain axis, portions of the brachial plexus, the anterior crural nerve, the vertebral and left femoral arteries, and kidney were secured for microscopical examination. The pieces of the nervous system were hardened by Marina's method, stained by the Weigert-Vassale and Van Gieson's methods for study of the fibers, and by Nissl's method, and by thionin, for cell study. The other organs were hardened in alcohol and stained with hematoxylin and eosin.

In the sections of the cortex from the right side there was some increase in the neuroglia, and in the softened areas many cells had disappeared, but in those which remained there were no characteristic changes. Throughout the brain axis and in the upper part of the cervical cord the pyramidal tract from the

right hemisphere was degenerated, as were also most of the cerebro-pontine fibers, both those from the frontal and those from the temporal and occipital lobes. In the medulla and cord the cells of the various nerve nuclei appeared to be normal, though they were, perhaps, somewhat fewer in number on the left (the paralyzed) side. The sections from the brachial plexus and anterior crural nerve showed nothing abnormal. The vertebral arteries were somewhat narrowed in caliber, especially the left, the intima being thickened. The femoral was normal. The kidneys showed slight interstitial nephritis.

It is evident that the pathological findings do not differ from those met with in ordinary cases of hemiplegia, and do not directly explain the occurrence of the edema.

The condition of edema in hemiplegia is a rare one. Gowers¹ states that in hemiplegia slight edema is often present in the paralyzed limbs, and that it may be of greater extent, but mentions no example of its occurrence in any such degree as in this case. On searching the recent literature, I have only been able to find reports of three such cases, one by Hare,² of Philadelphia; one by Preobrajensky,³ of Moscow; and one by Gilbert and Garnier,⁴ of Paris. In the first of these the right arm alone was affected. In the second there was extensive edema, and eventually gangrene of the paralyzed extremities, but the case was complicated by aneurism of the aorta and thrombosis in the vessels of the hand and foot. The last case was exhibited before the Société de Biologie, of Paris, as one of hemiplegia with greatly swollen hand, presenting a condition not to be distinguished from the so-called "succulent hand," declared by Marinesco to be pathognomonic of syringomyelia. The patient was still alive when the observation was published. In the first two cases the autopsy showed, in common with the one reported here, that large areas of the brain were affected. There is no satisfactory explanation of the production of the localized edema, however.

Von Monakow⁵ makes bare mention of edema in hemiplegia, though he says that in the early stage there is a

¹Gowers, "Diseases of the Nervous System."

²Hare; *Journal of Nervous and Mental Disease*, 1898, p. 189.

³Preobrajensky, *Memoires médicaux de Moscou*, 1896, No. 16. Ref. in *Révue neurologique*, 1897, p. 73.

⁴Gilbert et Garnier; *Comptes rendus de la Société de Biologie* 1897.

⁵Von Monakow; "Gehirnpathologie."

tendency to slight swelling and increased warmth of the paralyzed limbs, while in the chronic stage coldness and lividity, with glossiness of the skin, are the rule. In speaking of other trophic disturbances in hemiplegia (particularly of muscular atrophy), however, he calls attention to the fact that in every case the brain lesion has been quite extensive, and this seems to have been the rule also in the cases of edema in hemiplegia which have been reported.

The circulation of both blood and lymph is favored by the action of the muscles; hence, loss of power in the muscles on the one hand, and failure of vasomotor control on the other, would seem to be responsible for the production of stasis and edema on the paralyzed side. As to why this so rarely occurs, however, we have no definite information.

147. ANALGÉSIE ÉPIGASTRIQUE PROFONDE CHEZ LES TABÉTIQUES (Deep Epigastric Analgesia in Tabetics). Prof. Pitres (Journal de Médecine, 70, 1899, p. 60).

Pitres has added a new sign of some importance to the symptomatology of tabes. The sensation of anguish, leading sometimes to unconsciousness, which follows a blow on the epigastrium is well recognized. But in certain pathological conditions, the deep epigastric sensibility, normally so acute, is completely absent. In cases of this kind one can make forcible pressure over the pit of the stomach, or strike the epigastrium violently with the fist or a hard body, without causing the patient any discomfort. Deep epigastric analgesia exists in a very high degree in a large number of cases of hysteria. This explains the indifference with which the convulsionnaires of Saint-Médard strike themselves on the stomach with stones or bars of heavy metal. This deep analgesia is not only found in hysteria, but is met in a certain number of cases of organic maladies of the nervous system, notably in general paralysis and in sclerosis, but in tabes its frequency is relatively great. In this last malady in particular, Pitres has found the epigastric sensibility very much diminished, twenty-two times in fifty cases, that is to say, in almost half the cases, and completely abolished nine times in fifty cases. This analgesia does not appear to be associated with any sensitive trouble, anesthesia of the skin, loss of abdominal reflex or gastric crises. The only functional trouble with which it appears the most habitually associated, is loss of hunger sensation. It is difficult to explain this loss of sensation—and also the analgesia of the testicles, breasts and anesthesia of the bladder, urethra and rectum. It is probable that these conditions are due to a visceral neuritis analogous to the peripheral neuritis which causes in tabes certain disturbances of the special or general senses.

FREEMAN.

TUMOR OF THE OBLONGATA PRESENTING ATAXIA AND
ASTEREOGNOSIS AS THE MOST PROMI-
NENT EARLY SYMPTOMS.*

By F. X. DERCUM, A.M., M.D.,

CLINICAL PROFESSOR OF NERVOUS DISEASES, JEFFERSON MEDICAL COLLEGE;
NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

The following case, both because of the infrequency of tumors involving the medulla and because of certain unique features in the symptomatology, deserves to be placed on record.

A.M.; aged 26; female; married; first came under the observation of the writer on September 19, 1898. She complained more especially of inability to properly use the right arm.

Family History.—The family history was negative, especially as regards nervous diseases. The father and mother of the patient were living and well. Two brothers and one sister were living and well. No deaths had occurred in the generation to which the patient belonged.

Personal History.—The patient could not remember any illness of moment occurring during childhood. Menstruated at fourteen and continued to be perfectly well up to fifteen years of age, when she was attacked by pain in the right side of the neck. The neck became very stiff and the pain gradually increased until it attained a very high degree of severity. Both the rigidity and the pain resisted for the time all efforts at treatment and persisted for about a year. The pain then gradually subsided and finally disappeared and with it the rigidity. Subsequent to this attack the patient continued to be entirely well until about two years ago when pain again made its appearance in the right side of the neck. As far as she can remember, it was in exactly the same situation as before, and just as on the former occasion it was attended by stiffness of the neck. This attack lasted six months. However, unlike the former attack, neither the pain nor the rigidity, although they somewhat improved, ever entirely disappeared. There has been ever since more or less pain in the right side of the neck. This condition persisted without change until three months ago, when the patient began to feel in addition some numbness in the fingers and thumb of the right hand. A little later the fingers and hand began to feel stiff and awkward and she be-

* Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

gan to drop various articles which she attempted to hold. A sense of stiffness and of weight subsequently spread over the hand up to the shoulder, involving the entire arm. She has not been able to play the piano for about three months. She noticed also three months ago that she could not write as well as formerly, and this difficulty has steadily increased.

The patient states that she was married at twenty years of age, that she has had two miscarriages, but no living children. There is no history of alcoholism.

Present Condition.—September 19, 1898. The patient states that her general health has failed somewhat; that her sleep has become disturbed; that her appetite has become poor; that the right arm has a "numb and dead feeling," and that this feeling extends from the tips of the fingers up as far as the right side of the neck, and that this numbness is associated with a sense of stiffness. She also states that her neck feels somewhat stiff at present, and that when she goes to move the head, her neck "catches her." In making this statement, she indicates the origins of the trapezius and sterno-mastoid muscles.

Station with eyes closed reveals a slightly exaggerated sway. In walking the patient betrays a barely perceptible awkwardness in the use of the right leg. Otherwise the gait is normal. She stands very well upon the left leg alone; stands upon the right leg alone, but makes a much greater effort at balancing. The right knee-jerk is exaggerated. There is also present a right ankle-clonus, but it rapidly dies out. The left knee-jerk is also exaggerated, but less markedly than the right. There is no ankle-clonus on the left side. The elbow and wrist jerks are present and somewhat pronounced, equally so upon both sides.

The right arm is moved freely in all directions at the shoulder joint, but with an appearance of effort and awkwardness. The arm is also readily flexed and extended at the elbow, but on attempting forcible flexion of the biceps the arm is found to be not as strong as its fellow. Movements of the wrist are performed in all directions, but the extensors are weaker than those of the left side. Grip of the right hand registers 22; left hand, 35. Movements of the fingers are executed freely, but like the movements of the arm, they are decidedly ataxic. The movements of the arm as a whole, as shown by efforts to touch the nose or chin with the eyes closed, are decidedly ataxic. The handwriting is very irregular and hardly legible. The movements of the left arm and hand are in every respect normal. Patient is right-handed.

Examination as to sensory changes reveals no anomalies in

the face, trunk, the left arm or either leg. In the right arm and hand a detailed examination reveals that the tactile sense, temperature-sense and the pressure-sense are well preserved. The pain-sense is slightly diminished over the forearm, hand and fingers. It is noticed also that the patient makes frequent errors of location of the tactile impressions. She also makes occasional errors in describing the position in which the arm, hand or fingers are placed. In testing her muscular sense by means of objects of various weights, it is found that the muscular sense of the right arm is greatly diminished or lost. It is now observed that the patient is also unable to recognize various objects placed in her hand when her eyes are closed, although she recognizes these objects promptly when placed in the left hand. In other words, there is complete loss of the stereognostic sense as regards the right hand. She can tell when a key or coin is placed in her hand, *i. e.*, she recognizes the presence of a foreign body, but is utterly unable to name or describe it. She can tell whether it is cold or warm and instantly recognizes its presence, but she cannot describe its shape or give any other information as to its physical qualities. In addition the right hand is very slightly tumid. This is noticeable in the fingers and thumb in which the wrinkles, especially of the middle and distal phalanges, are slightly less marked than those of the left hand. The hand as a whole looks somewhat smoother than the left.

Superficial or deep pressure over the spinal column fails to reveal any evidence of pain or tenderness. There is no inguinal, inframammary or other superficial area of hyperesthesia or tenderness. However, a patch of deep-seated tenderness, not marked, is discovered on the right side of the neck, immediately back of and over the upper third of the belly of the sterno-cleido-mastoid muscle. The patient states that this is the area in which she felt pain and stiffness in the neck both in the attack eleven years ago and the attack beginning two years ago. This pain sometimes spreads down the neck as far as the axilla. No tenderness is elicited over the brachial plexus or over the nerves of the arm. On asking the patient to move the head freely in all directions, it is found that the head is not rotated as freely as normally, either to the right or to the left, and also that the movement gives rise to "a catching pain." This is also true of the movements of flexion and extension. The limitations of the movements of the head and neck, while not marked, are unmistakable.

The electrical examination of the muscles of the right arm fails to reveal any reaction of degeneration. The muscles of the two arms respond equally; possibly those of the right arm re-

spond a trifle more readily than those of the left to the same minimal current.

There is no involvement of the face, tongue or soft palate. The patient states that there is no sensation of numbness of the legs, feet or toes, or of the left arm. There is no headache or any pain whatever save that in the side of the neck. The patient is markedly constipated. The sphincters are normal. The examination of the special senses reveals no anomalies. The pupils respond normally. The eye-grounds are perfectly normal. There is no contraction of the visual fields. These findings were confirmed by Dr. de Schweinitz, who reported in addition that the color-fields were normal save a slight contraction for green.

The urine is negative.

October 12.—Is brought by her physician, who reports that she is taking potassium iodide in ascending doses; at present thirty-five grains three times daily. For five days past she has also been receiving two drachms of mercurial inunction daily. No traces of iodism or mercurialization are present. The right hand registers by the dynamometer, 22; the left, 35. Ataxia of right arm possibly a little more marked. Other symptoms as before. No loss of cutaneous sensibility other than the slight degree noted at the previous examination.

October 29.—The patient now complains of pain in the *left* side of the neck and upper part of the *left* shoulder. A patch of deep-seated tenderness is now revealed on the *left* side of the neck, above the sterno-cleido-mastoid muscle, in a position corresponding to that in which the patch is observed on the right side. There is now also undoubted, though very slight, ataxia in the movements of the *left* arm. The patient also complains of numbness of the left hand, involving more especially the ring and little fingers and the ulnar side of the hand and forearm. In the left upper arm also there is a band-like sensation of tightness or constriction. No loss of the stereognostic sense can, however, be detected on this side. The other symptoms are as before. The patient is now unable to describe the position of the right arm and hand at all, and she volunteers the statement that she "sometimes loses her hand in the bed."

November 7.—Since November 3 has begun to drag the right foot. Sway with eyes closed is exaggerated. The patient is unable to stand upon the right leg alone. She is, however, able to describe the various positions in which the leg is placed. Both knee-jerks are greatly exaggerated, the right slightly more than the left. The stiffness of the neck is now much more marked than at any previous time. Rigidity is

noted upon attempting flexion, and especially extension. Since the last two or three days she is unable to extend the right arm at the shoulder. Loss of power in the right arm is now very pronounced. Flexion of the forearm is very feebly performed, and all the movements of the hand, while preserved, are feebler than before. Movements of the left arm not as readily performed as at last examination. This is due especially to weakness and to some slight increase in the ataxia noted before. The left hand now registers by the dynamometer 20, while the right hand registers 11.

Re-examined, November 14, at the patient's home. Patient is in bed. History since last visit at office is that there has been a gradual and increasing paralysis of the right arm and right leg, of the left arm, and finally of the left leg. No movement whatever can now be made in either arm. Very feeble movements can be made in the right leg at knee and toes. Movement in the left leg also excessively feeble, but a little better preserved than in the right. Elbow and biceps jerks preserved and equal on both sides. Right knee-jerk plus; left knee-jerk double plus. Feeble right ankle-clonus; left ankle-clonus pronounced and sustained. Cutaneous sensibility much lessened to all forms over both legs, especially over the right. Cutaneous sensibility also diminished over both arms, more especially over the right. Sensation now much diminished over the trunk. Over left half of the trunk, sensation is a little better preserved than over the right half. Sensation appears to be well preserved at the level of the clavicles. There is, however, no sharp line of demarkation. Impressions at the level of the nipples are perceived almost, but not quite, as readily as below the clavicles. Over the shoulders there is also a diminution of sensation, but not as marked as in the arms. Sensibility to all forms is well preserved over the neck, face and head. The pupils are equal and react to light. The eye-grounds are still normal. The patient complains of much stiffness in the back of the neck. On attempting to move the neck it is found that the rigidity noted in the former examination is more pronounced, and that attempts at passive movement give rise to pain. If the patient be raised without supporting the head, the latter falls backward or it sinks forward on the chest, as though the muscles of the cervical region were too weak to properly hold it up. Attempts at blowing or at straining produce no movement of the abdominal wall. The breathing is almost entirely performed by the upper portion of the thorax; no movement of the diaphragm can be detected. The respiration is 26; pulse 96. The face is dusky and flushed. There is occasional strangling on attempting to swallow food. Solid

food cannot be swallowed at all. The temperature is normal. There is at present retention of urine and marked constipation.

November 15th, 4 P. M.—Patient has not slept all night, but fell asleep at 12 o'clock noon to-day and woke about 3. During the sleep there was a rattling in the throat, probably produced by the relaxation of the uvula. There is no motion of the alæ of the nose in breathing. The tongue is protruded straight. The face is symmetrical. The pupils are normal. The patient is cheerful and replies to questions put to her in short phrases. Condition about the same as at last examination, except that paralysis of the trunk and limbs is more pronounced. Constipation and retention of urine as yesterday.

Examined, November 16th, 4.30 o'clock.—The paralysis and sensory losses noted on previous occasions have become more pronounced. No sharp lines of demarkation exist between area of preserved sensation in the neck and the area of diminished sensation in the trunk. The sensation below the clavical and the regions of the breasts, for instance, is impaired, but passes by gradual transition to normal sensation when the neck is reached. Obstinate constipation continues. Temperature normal. Pulse 108.

Examined, November 17th, 8.45 o'clock.—Symptoms more pronounced. Sensory loss now involves the neck, extending up to the level of the lower jaw. Sensation appears to be normal in the upper half of the cheek and forehead. Along the sides of the head and back part of the head sensation appears to be very much impaired. The pupils are a trifle dilated. A rise of temperature, 101° , has been noted since morning. Symptoms otherwise unchanged. Pulse 112.

Examined, November 18th, 4 P. M.—Face livid, bluish red, somewhat tumid. Temperature 105° . Pulse 120. Respiration hurried, feeble and shallow. Pupils slightly more dilated than at previous examination. Patient much confused mentally; delirious. Does not recognize persons about her. Is evidently dying.

Died November 18th, 4.30 P. M.

Autopsy November 20th, 1898, 9 A. M.

The calvarium presents no abnormality. The dura is not adherent or thickened. Some slight deposit of dark red granular material is found on the pia matter upon either side of the margin of the longitudinal fissure. It is easily scraped off with the finger-nail, and is more especially noticeable in the right hemisphere, upon the central convolutions, but is not limited to these areas. Subsequent microscopical examination revealed this granular matter to be altered blood pigment, and also that

the pia was infiltrated with it. The pia and cortex are in every other respect normal. On removing the brain from the cranium a large irregular mass, springing from the occipital bone on the right side of the junction of the pons and medulla oblongata, and forming a slight depression in the right lobe of the cerebellum, is revealed. The right vertebral artery is occluded from pressure. The medulla oblongata is markedly distorted, the greatest distortion occurring at the lower part of the inferior olive. The right olive is elongated antero-posteriorly, while the left is more nearly of the normal shape. A transverse section of the medulla oblongata in its largest portion antero-posteriorly measures 2.2 cm. and laterally 1.3 cm.

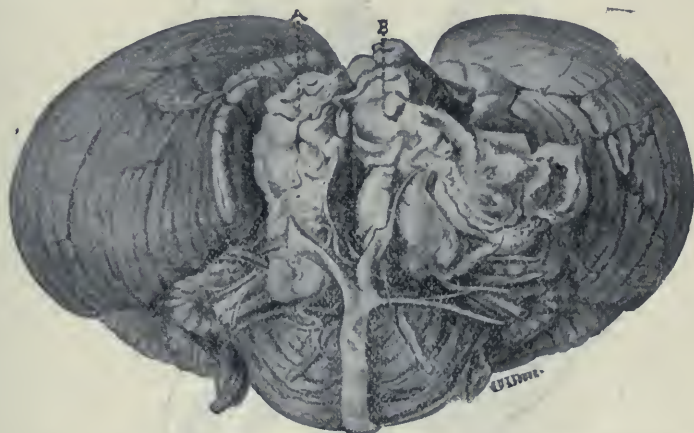


Fig. I.—A, medulla oblongata; B, tumor pushing the medulla oblongata to the left.

The distortion caused a projection of the right anterior pyramid beyond the left, and of the right posterior columns posteriorly beyond the left. The greatest pressure appeared to have been exerted on the median portion of the right side of the medulla oblongata, although the entire right side showed the effects of pressure. The pia of the medulla oblongata was infiltrated with altered blood pigment as the portion of the cortical pia already described.

Microscopical examination failed to reveal any degeneration by Weigert's hematoxylin method or the carmine stain, but the employment of the Marchi stain gave more successful results. Sections stained by the Marchi method show a degeneration of myelin sheaths, which is most marked just below the area of greatest compression; it is therefore most distinctly seen at the lower part of the motor decussation. The posterior

and lateral columns at the level of the lower part of the motor decussation are much degenerated, and the left side appears to be as much affected as the right. The left anterior pyramid reveals a little more degeneration than the right. Distinct degeneration is seen in the area of the right direct cerebellar tract while in the corresponding area on the left side no degeneration can be seen. The degeneration seen at the motor decussation does not extend very far downward or upward. At the



Fig. II.—Section from the medulla oblongata at the point of greatest compression. The lower olive on the side of the tumor is elongated from pressure.

junction of the pons and medulla all evidences of the degeneration have disappeared. Similarly sections taken from the upper cervical cord and stained by the Marchi method show no alteration. It should be added, however, that the piece of the cervical cord immediately joining the medulla was not removed at the autopsy. The alteration of the myelin at the motor decussation, as seen by the Marchi method, is possibly

the same as that produced after death when the nervous tissues are handled carelessly or injured, but in this case the lesion seems undoubtedly to have been the result of pressure *intra vitam*.

Fibers from a posterior lumbar root in the upper part of the lumbar swelling, stained in the fresh state with osmic acid, and teased, appear to be normal. This examination was made on account of the frequently reported degeneration of the posterior roots in brain tumor.

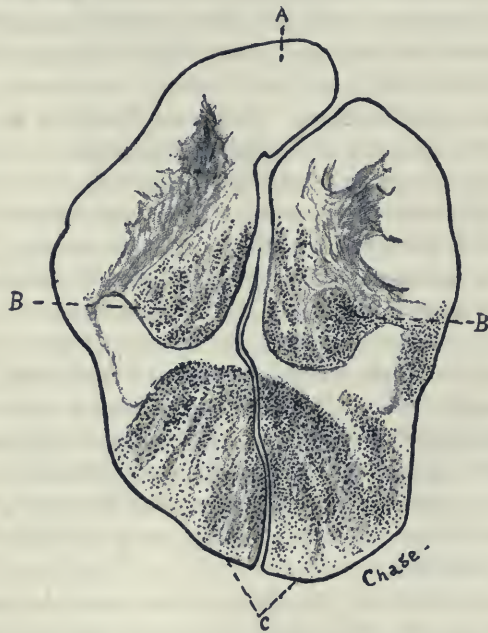


Fig. III.—Section from the lower part of the medulla oblongata. A, anterior pyramid; BB, motor decussation; C, posterior columns. The dots represent the degenerated fibers shown by the Marchi method.

Sections from the lumbar region, stained by the Marchi method, show no degeneration of the posterior roots.

Microscopical examination of the tumor showed it to be a chondrosarcoma.

It is interesting in connection with these findings to briefly review the early symptoms of the case. It will be remembered that these consisted especially of ataxia of the right arm, some weakness of the muscles, loss of the muscular sense, loss of the

sense of position of the limb, slight diminution of the pain-sense and some disturbance of the sense of tactile localization. The other cutaneous sensibilities, tactile sense, temperature-sense and pressure-sense were normal. There was also very slight ataxia of the right leg. It is extremely probable that the motor weakness is directly related to the pressure upon the right motor tract, immediately below the decussation. The cutaneous sensory disturbances are in all probability to be ascribed to pressure upon the right posterior columns. It is also probable that the ataxia, loss of muscular sense and sense of position of the limb, are to be referred to pressure upon the right direct cerebellar tract. This tract, it will be remembered, as seen in the sections, is decidedly degenerated. It is further probable that the symptom of astereognosis is to be ascribed to the partial sensory losses which interfered with the complete perception of the qualities of the objects handled, and thus prevented a complete mental picture being formed in the cortex. Astereognosis has never before been observed as a symptom in tumor of the medulla. In fact, in all of the cases thus far reported in which astereognosis has been noted, a cerebral lesion was either definitely determined or the symptoms pointed unmistakably to the cerebrum as the seat of disease. Thus Wernicke's,¹ Monakow's,² Burr's,³ and Dubbers',⁴ cases resulted from trauma of the head, while Olmstead's,⁵ Sailer's,⁶ Williamson's,⁷ and Vorster's⁸ cases were all due to various lesions of the brain.

It is not within the province of this paper to enter into a detailed discussion of the subject of astereognosis. This was investigated by Hoffmann⁹ in 1883, who studied the subject

¹ Wernicke, "Arbeiten aus der psychiatrischen Klinik, in Breslau," Leipzig, 1895.—Also Monakow, "Gehirnpathologie," Nothnagel, Wien, 1897, p. 409.

² Monakow, "Gehirnpathologie," Nothnagel, Wien, 1897, pp. 410 and 411.

³ Burr, *Journ. Nervous and Mental Disease*, Jan., 1898.

⁴ Dubbers, *Neurologisches Centralblatt*, 1897, p. 61.

⁵ Olmstead, *Journ. Nervous and Mental Disease*, Nov., 1898.

⁶ Sailer, *Journ. Nervous and Mental Disease*, March, 1899, p. 161.

⁷ Williamson, *British Med. Journ.*, 1897, II, p. 787.

⁸ Vorster, *Archiv. f. Psych.*, Bd. XXX, No. 2, 1898, p. 341.

⁹ Herman Hoffmann, "Stereognostische Versuche," Inaug. Dissertation, Strassburg, Konstanz, 1883.

quite extensively. Since Hoffmann's thesis appeared merely a limited number of cases presenting astereognosis have been reported. Recently, however, Sailer¹⁰ has again written upon the subject. According to Hoffmann a large number of factors enter into stereognostic perception; for instance, the tactile sense, the sense of localization of tactile impression, the space- or distance-sense (*Raumsinn*), the pressure-sense, the muscular sense, the temperature-sense, the pain-sense, the sense or knowledge of the position of the fingers to each other and to the hand, the recognition of passive movements and the knowledge of active movements. It can readily be seen that the faculty of perceiving objects by means of the hand is an exceedingly complex one. The various impressions received from all of the sources enumerated by Hoffmann, and perhaps others, are combined in the cortex to give rise to a mental picture of the object felt. It can readily be understood that if there be an elision of some of these impressions, this mental picture cannot be formed. The relative importance of the various impressions has as yet not been satisfactorily determined. Hoffmann states that the temperature-sense, the pain-sense, the tactile sense, the sense of position of the limb and the appreciation of the weight of bodies may be preserved, although the stereognostic sense is lost. The ability to appreciate the distance between two points, the sense of pressure, the recognition of the movements of the joints, and the recognition of the relative position of the fingers are, in Hoffmann's judgment, more important, for he says that in not one of his cases in which any one of these senses was preserved was the stereognostic sense lost. A diminution was, however, several times noted by him with almost complete integrity of one of these senses, and even at times of two. He states, on the other hand, that each one of these senses may be diminished to a marked degree without the stereognostic sense suffering in like degree, and further that the involvement of all of these senses does not necessarily involve the stereognostic sense. As regards the faculty of appreciating distance on the cutaneous surface, he says that the stereognostic sense may be entirely intact, although this faculty

¹⁰ Loc. cit.

be diminished, and on the other hand may suffer severely when this faculty is normal. Again, complete loss of this faculty is not followed by absolute loss of the stereognostic sense. He also found that the stereognostic sense may be intact with greatly impaired pressure-sense, and with perfectly preserved pressure-sense may be lost. It can readily be seen from Hoffmann's results that the various elements entering into stereognostic perception are of variable value, and are possibly to some extent interchangeable. No one sense, but a combination of many, is necessary to stereognostic perception. Sailer, correctly it appears to the writer, gives more importance than does Hoffmann to the tactile-localization sense.

In the case here reported there was, in addition to ataxia, distinct loss of the muscular sense, loss of the sense of the position of the limb, diminution of the pain-sense and disorder of the localization of tactile impressions. It can readily be understood, therefore, that although the tactile sense, the pressure-sense and the temperature-sense were well preserved, the faculty of perceiving foreign bodies was lost.

It is exceedingly probable, as already stated, that the ataxia, loss of the muscular sense and of the sense of position of the limb are to be referred to the degeneration of the direct cerebellar tract. It is not improbable also that the disorder of sensory location was due to pressure upon the right posterior columns, the interference of function, however, being so slight as merely to produce *confusion* of tactile impressions instead of tactile loss. The degenerations noted in the motor tracts and posterior columns of both sides are probably of very recent origin. This is proven not only by the failure of the Weigert method and the carmine stain to demonstrate them, but also by the history of the case. It was merely twenty days before death that any symptoms were noted referable to the *left* arm, and then they were so slight as to be barely appreciable. It was not until fifteen days before death that these symptoms became at all distinct, and not until five or six days before death that the patient became markedly paraplegic from the neck down.

In its general features, especially as regards the pain and rigidity of the neck and exaggeration of the deep reflexes, the case recalls the tumor of the upper cervical cord reported by

Dr. Collins;¹¹ in its special features it recalls the case of glioma of the medulla oblongata reported by Dr. Collins,¹² in which there was paresis, distinct ataxia, exaggerated tendon-reflexes and ankle-clonus, more particularly of the left side, together with loss of the pain-sense and with preservation of the temperature- and tactile sense. In Dr. Collins' case, like the one here reported, there was also no optic neuritis. The feature of ataxia of the arm and slight ataxia of the leg recalls the case reported by Higier,¹³ which was probably one of thrombotic softening of the medulla; the patient presented ataxia of the left extremities. The lesion was apparently due to syphilis, improved under specific treatment, and was, therefore, not verified by autopsy.

The symptoms, however, leave no doubt as to the probable situation of the lesion. The case here reported also recalls that of Wenhardt¹⁴ of myxochondrosarcoma of the base of the cranium compressing the left hemisphere of the cerebellum, the medulla and pons, and giving rise to pain in the back of the neck, paroxysmal pains in the head, impaired movement of the head, hemiatrophy of the tongue, difficulty of swallowing and exaggerated knee-jerks. In Wenhardt's case there were, however, no sensory disturbances. Optic neuritis also was absent. It further recalls the case reported by Eskridge,¹⁵ in which there was an intradural sarcoma extending from the upper portion of the cervical cord into the skull through the foramen magnum. In addition to paraplegia there was present exaggeration of the knee-jerks, loss of the tactile and temperature-sense below the inferior maxilla, and in addition to these changes also loss of the pain-sense in the arms and legs; there was no optic neuritis.

On October 24th, 1898, the writer presented this case *intra vitam* before the Philadelphia Neurological Society. The possibility of a lesion of the cortex, probably in the posterior portion of the superior parietal lobule, was discussed. The writer, however, hesitated to accept the diagnosis of cortical lesion

¹¹ Collins, Medical News, 1897, No. II, p. 48.

¹² Collins, Zeitschr. f. Nervenheilk., X, p. 467.

¹³ Higier, Zeitschr. f. Nervenheilk., 1898, 13, p. 316.

¹⁴ Wenhardt, Neurolog. Centralbl., 1898, p. 541.

¹⁵ Eskridge, Medical News, 1897, No. II, p. 402.

because of the slight ataxia of the *left* arm, which at this time had begun to make its appearance, and also because of the rigidity of the neck. The fact that the iodides and mercurials were so well tolerated suggested, as a possibility, a multiple lesion, one cortical and the other cervical, both due to syphilis. The subsequent progress of the case, however, left no doubt as to a lesion being present in the medulla or cord high up, and this view was shown to be correct by autopsy. The new and striking feature of the case is undoubtedly the astereognosis, and this symptom at first sight suggested, though erroneously, a lesion of the brain.

The writer is under obligations to Dr. William G. Spiller for assistance at the autopsy and for the microscopical work upon the specimens, done in the William Pepper Laboratory of Clinical Medicine.

148. A CASE OF ACUTE TETANUS TREATED BY INTRA-CEREBRAL INJECTIONS OF ANTITOXIN: RECOVERY. William F. Gibb. (*British Medical Journal*, No. 1998, page 895, April 15th, 1899.)

The patient was a lad of thirteen years, and the period of incubation sixteen or seventeen days. The first day of the disease he received ten grains of chloral every four hours. At two P. M. on the second day, 10 c.c. of antitetanic serum were administered under the skin, and four hours later, under chloroform anesthesia 8 c.c. were injected into each frontal lobe of the brain and 14 c.c. subcutaneously. At 10.30 he received 10 c.c. under the skin. On the evening of the third day, 10 c.c. were administered hypodermically. During the fourth day the patient was a little better, and on the fifth day, although the tetanic symptoms were about the same, he was quieter. In the evening 10 c.c. of serum were injected into each frontal lobe. The sixth day showed practically no improvement, and he received 20 c.c. hypodermically. The same dose was given the next (seventh) day, and on the eighth day he was quiet and had 20 c.c. of serum in the evening, but the following day he seemed worse and was given 15 c.c. by intracerebral injection. For two days he remained in about the same condition, and then began to improve, making a slow recovery.

The case is open to objection as evidence in support of intracerebral injections, because the serum was given freely under the skin, and because of the comparatively long period of incubation, the prognosis being comparatively favorable under any treatment when the disease does not declare itself within two weeks after the inoculation.

PATRICK.

TUMOR OF THE PITUITARY BODY*.

ABSTRACT.

A paper with this title was read by Dr. G. L. Walton, in conjunction with Dr. Cheney, of Boston.

Tumors of this region have acquired a new interest since the demonstration of acromegaly by Marie. Few authorities now deny that a relation exists between disease of the pituitary gland and acromegaly, though arguments are still adduced to show that the diseased gland represents a result rather than the cause of the general perversion of function.

The interest in these cases is now three-fold: (1) in their bearing upon the anatomy of the chiasm and optic tract; (2) in their symptomatology as bearing upon early diagnosis, and (3) in their pathology, more particularly with reference to the destruction of glandular function.

(1) The optic chiasm is the first important body affected by the pressure. The tumor may extend upwards, either in front of the chiasm or behind it, or both, causing amblyopia and various forms of field restriction. Both temporal fields are often lost; sometimes one eye is completely blind while the other shows loss of temporal field, and in Leszynsky's case, as in ours, one temporal field alone was affected at a certain stage. Homonymous hemianopsia is sometimes found (as in our case at a later stage) showing pressure on the optic tract behind the chiasm. (2) The symptoms, apart from the function of the gland itself, are headache, visual field affection, optic atrophy, vertigo, disturbance of motion, spastic and paretic (pressure on the *crura cerebri*), disturbance of speech, disordered pupils, paralysis of ocular muscles, and diabetes (mellitus and insipidus). To these symptoms must be added those of acromegaly, symptoms less marked when the disorder appears in adult life. Pechkranz has suggested that acromegalic symptoms dating from childhood may be due to congenital anomalous structure of the pituitary body. (3) The commonest form of new growth partakes of the nature of adenoma. In some cases

* Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15 1899.

the diagnosis between adenoma and sarcoma is difficult. Hip-pel asserts that all these growths start from the anterior lobe. Burr and Riesman argue that the lack of acromegaly in certain cases of tumor of the hypophysis points to the retention of a certain amount of healthy gland tissue.

CASE.

A physician, 26 years of age, first consulted Dr. Cheney, July 20, 1895, for migraine dating from early life, with temporal hemianopsia—a large man with rather heavy gait, large extremities and features, including broad nose and very thick lips, large tongue, large jaws with separation of teeth. He complained of *asthenopia*. The vision in the right eye was .7; left, normal. Fundus examination negative. Headaches became more frequent, and permanent temporal hemianopsia appeared in the left eye. Optic atrophy ensued. In May, 1896, the vision in the right eye was reduced to .2, in the left eye to .5. The field for form and color in the right eye was unaffected. Somnolence and mental apathy appeared and increased, though he was still able to practise. In January, 1897, Dr. Derby saw the case. At this time the nasal field of the right eye had disappeared (homonymous hemianopsia). In September of 1897 there was general physical weakness, and at this time color sense was absent in the otherwise normal temporal field of the right eye. His speech was slow. The first of October he gave up work. The headaches were becoming more frequent, somnolence and apathy increasing. Lack of sexual power was complained of throughout the history of the case. Patient died in December, 1897.

PATHOLOGICAL EXAMINATION BY DR. MALLORY.

Tumor 2.7 cm. by 3.5 cm. by 3 cm., causing the disappearance of the olivary process and portions of the sella turcica. Growth soft and granular, dark gray in color, with soft translucent light grayish masses resembling cartilage, but softer (colloid). Soft septa extended into the growth from the fibrous capsule. Growth consists mainly of delicate blood vessels surrounded by walls of round cells generally epithelioid in type, the darker areas due to hemorrhage. Along one of the septa

extend narrow compressed tubules, some of the cells of which tend to take eosin stain like those of the normal pituitary gland after fixation in Zenker's fluid, some of these tubules dilated and containing colloid material.

Diagnosis: *Perithelial angiosarcoma* (endothelioma).

The order of optic symptoms points to involvement of the right optic tract at its junction with the chiasm, the lesion extending to involve the whole optic tract so as to produce finally hemianopsia. The amblyopia points to affection of the macular fibers. The achromatopsia shows that color-sense may be impaired by peripheral as well as by central lesion. The writers are strongly inclined to think that the peculiarities in growth (large features, etc.), though not sufficiently marked to be deemed abnormal, pointed to the congenital defect of the gland structure forming the basis of the new growth which appeared later in life. The lack of added symptoms of acromegaly was partly due to the patient having reached adult life, and partly to the preservation of an area of comparatively healthy gland tissue.

CONCLUSIONS.

1. Congenital peculiarities in growth resembling those of acromegaly, but occurring in otherwise healthy individuals, may point to structural defect of the pituitary gland, a defect sometimes furnishing the starting point for new growth later in life.
2. The occurrence of pituitary tumor without definite symptoms of acromegaly does not necessarily disprove a connection between this organ and this disease, for the persistence of even a small amount of healthy gland tissue is sufficient to carry on fairly the function of the pituitary body.
3. The combination of general symptoms of new growth with optic atrophy and loss of temporal field of vision makes the diagnosis of pituitary tumor almost certain.
4. Hemiachromatopsia is not necessarily of central origin.

DISCUSSION.

Dr. W. G. Spiller thought that the intense degeneration of nerve fibers in the medulla oblongata, without secondary ascending or descending degeneration, in Dr. Dercum's case was worthy of comment. The nerve fibers had not been cut,

and the degeneration was evidently caused by the pressure of the tumor. He was not aware that such a condition had often been seen in cases of tumor of the central nervous system, and he regarded it as similar to that in the periaxillary neuritis of Gombault. It seemed probable that the axis cylinders had not been seriously injured and that the degeneration was limited to the medullary sheaths. Secondary degeneration would probably have occurred if the patient had lived longer.

Dr. Langdon said, in regard to the case reported by Dr. Dercum and the relation of the growth to the impairment of the stereognostic sense, that it seemed to him that this sense was formed by the fusing of a great many sensations. One of these fused elements was probably in his opinion the labyrinthine sense which gives us knowledge of our general position in space, and there was no reason why, in his estimation, it should not give us an idea of the position of the separate *parts* of our bodies in space. The location of the lesion was significant in Dr. Dercum's case, as it involved the labyrinthine fibers of the oblongata, and it would have been of interest to test the functions of the internal ear in such a case.

Dr. Dercum replied that the ears of his patient had been examined. She had no abnormality of hearing, no vertigo, and there was nothing to suggest the involvement of the fibers coming from the labyrinth.

Dr. Walton said that in illustration of the general lack of appreciation of the fundamental symptoms accompanying pituitary tumors, he had noticed recently the abstract of a case presented to the Ophthalmological Congress of Paris as one of meningitis or new growth of unknown seat and character, except that it involved the chiasm. The patient was a woman of fifty-eight years, with bitemporal loss of the visual fields, optic atrophy, loss of vision to one-third and one-half of normal, headache and somnolence. This case, if correctly reported, presented therefore, the essential diagnostic features of tumor of the pituitary body.

INTERESTING HYSTERICAL PHENOMENA—A TRANSFER OF TACTILE TO VISUAL SENSATIONS.*

BY FRANK R. FRY, M.D., ST. LOUIS, MO.

The subject in whom the following experiments were produced was a hysterical girl, aged 14 years. I shall not go into the particulars of her case at this time except to enumerate a few data in order to give an idea of the character of the subject and her condition. Before the time that my examinations were made, January, February and March, 1898, she had been under the observation of Dr. Geo. M. Tuttle, who felt secure in a diagnosis of ulcer of the stomach on account of hemorrhage, pains, etc., in fact, typical symptoms with subsequent exhaustion. In this exhausted condition she had repeated attacks of delirium, alternating with more quiet hallucinatory states. She had frequently hemiopic hallucination and monocular diplopia. Soon after coming under my observation there appeared a complete anesthesia and paralysis of the whole right side. She was anesthetic to touch, temperature and pain tests made in the usual way.

Knowing of the Binet experiments I had no difficulty in reproducing them in this girl. I directed her to look at the wall (a plain white surface) on the left side of her bed and to tell me what kind of *objects* she would see there. I then traced on the anesthetic (right) arm and forearm various simple geometric figures: triangles, squares, circles, etc. She saw them distinctly on the wall, naming them without hesitation from the first. Letters and numerals carefully traced were recognized in the same way. The tracings were lightly made with the fingernail or an ordinary lead pencil not much sharpened. When directed to look at a colored screen during the tracing she always saw the figures surrounded by the color complementary to the one before her, *i. e.*, while looking at the blue screen she saw the figures in a red field and *vice versa*. Only blue and red screens were used, but other tests revealed a perversion of color perception.

* Read by title at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

Simple objects placed in the hand (the anesthetic hand), and the hand closed upon them, were seen upon the screen, *e. g.*, an opened match box. A silver dollar was recognized as a disk. A handkerchief was tied about the wrist. She immediately announced on the screen "a dark arm and hand with a dark cloth tied around it." Other tests quite as interesting were made. But I have related enough to draw attention to an interesting hysterical phenomenon, namely, that while the subject could not recognize in the ordinary way certain sensory impressions (tactile), she could interpret them by the aid of another sensory function (visual). The experiment of transferring by suggestion a hysterical anesthesia from one area to another is a familiar one. What I have just described is similar, and I have implied as much in my title, but it is not exactly parallel. In the latter case a more complicated psychic process takes place.

I saw my friend, Dr. Henry Hermann, of St. Louis, producing these phenomena ten years ago in a girl of about the same age as my patient. In the St. Louis *Polyclinic*, August, 1889, he made an extract from certain observations of Dr. Alfred Binet, of Paris, who had studied the phenomena in twelve subjects. I do not believe the matter has been much noticed, otherwise there would have been more reference to something so interesting and instructive as this is. It has seemed to be a difficult matter to explain well the difference between hysterical and organic anesthesia. Some authors after considerable detail of description fail to convey an idea of what may be considered the essential points of this difference. The above seem to me to be tangible data bearing on the subject. The object of this paper, however, is not to discuss the phenomena, but to prompt others to reproduce them in suitable subjects and thereby verify them.

It may not be out of place to state that great care was used in conducting these experiments. The nurse was carefully cautioned; and only she and persons who understood the necessity of caution were admitted to the room when the examinations were being made. Our patient never knew at any time that the tracings were made upon the arm, or that this member was used in any way in the process. She was curious

to understand how the pictures were produced. She constantly besought the nurse for an explanation, who finally told her that a small magic lantern was used for the purpose. She then frequently asked me when I would allow her to see the lantern. She was an intelligent and tractable child, a very good subject for this kind of work.

Drs. H. Hermann, Given Campbell and M. A. Bliss, all familiar with neurological work, witnessed the examinations. Subsequently the patient was under the care of Dr. E. C. Runge at the City Insane Asylum. Her case furnished many interesting features which I hope will be discussed by him at some future time.

149. UN CAS DE TREMBLEMENT SEGMENTAIRE DANS LA SCLEROSE EN PLAQUES (A Case of Limited Tremor in Multiple Sclerosis). Grasset (*Revue Neurologique*, 7, 1899, p. 270).

In this case of a woman of forty years of age in whom there was an undoubted multiple sclerosis, with spastic gait, scanning speech and intention tremor, there existed an intention tremor of the right upper extremity. It presented all of the characteristics of an intention tremor of this disease, except that it was limited to the movements of the fingers on the hand and the hand on the upper arm. It appeared in its characteristic manner when the classical effort to drink water was attempted. The fingers and wrist showed the changes in motion, spilling the water, but the upper arm and body were perfectly quiet, though normally having some amount of tremor. JELLIFFE.

150. DUE CASI D'IDIOZIA MIXEDEMATOSA (Two Cases of Myxedematous Idiocy). E. Tanzi (*Rivista di Patologia nervosa e mentale*, 4, 1899, p. 145).

The author here describes and figures in a very satisfactory manner two very interesting cases of myxedematous idiocy. One was in the person of a girl of fifteen years of age, the other in a male of twenty-two. Both of them presented the characteristic cutaneous dystrophies, with arrest of development, skeletal, sexual and mental. JELLIFFE.

NOTES ON THE ARRANGEMENT AND FUNCTION OF THE CELL GROUPS IN THE SACRAL REGION OF THE SPINAL CORD.*

By B. ONUF (ONUFROWICZ), M.D.,

ASSOCIATE IN PATHOLOGY AT THE PATHOLOGICAL INSTITUTE OF THE NEW YORK STATE HOSPITALS; LATE LECTURER ON NERVOUS AND MENTAL DISEASES IN THE NEW YORK POLYCLINIC; ASSISTANT NEUROLOGIST TO ST. CATHERINE'S HOSPITAL.

ABSTRACT.

The study of the group arrangement of the nerve cells of the spinal cord has been a rather neglected subject. This refers quite especially to the sacral region, and yet this part of the spinal cord presents so striking peculiarities in the grouping of its nerve cells that it is strange that so little attention has been paid to them. Of the few authors who have contributed to this subject Waldeyer¹ takes an important place. In his classical work on the spinal cord of the gorilla he refers extensively to the distribution of the cell groups in the sacral region, but he says nothing in regard to their functions. Recently Müller in a monograph issued from Strümpell's clinic² has given suggestions concerning the function of a certain cell column ("vegetative cell column") in the middle sacral region, which suggestions partly coincide with those to be brought out in this paper. However, he touches upon this point more as upon a side issue and not in a sufficiently exhaustive manner. Of recent authors I further mention Van Gehuchten and de Buck,³ and Sano,⁴ who from a study of the chromatolytic changes occurring in anterior horn cells of the lumbar and sacral regions after amputations, have formulated definite conclusions regarding the functions of certain cell groups of the lumbo-sacral region. I shall have occasion later on to return to this point, and shall now give briefly the results of my researches which were conducted at the Pathological Institute of the N. Y. State

* Read before the American Neurological Association, June 15, 1899.

¹Waldeyer: "Das Gorillarückenmark." Abhandlungen der Berliner Academie, 1886.

²Müller: Deutsche Zeitsch. für Nervenheilkunde, 1898, Vol. 14, p. 1.

³Van Gehuchten and de Buck: Journal de neurologie et d'hypnologie, 1898, p. 94.

⁴Sano: Journal de neurologie et d'hypnologie, 1897, pp. 253 and 274.

Hospitals, and a more extensive report of which will appear in the *Archives of Neurology and Psychopathology*.

First some remarks regarding the gross structure of the gray matter of the sacral region:

In studying the sacral portion of the spinal cord from above downwards, we find that at the level of the second sacral segment the configuration of the gray matter undergoes rapid and marked changes, to which Waldeyer¹ has already called attention. They consist:

I. In a remarkable widening, in dorso-ventral direction, of the gray commissure in general, and of that part of it in particular which lies dorsad of the central canal. This latter condition, namely, the widening of the retrocentral part of the commissure is due to

II. A coalescence of the bases of the posterior horns; therefore the non-united remainder of each posterior horn appears very short, consisting only of the head and a small neck.

III. A very marked reduction in the size of the anterior horns, chiefly in dorso-ventral direction.

IV. Widening of the central canal in dorso-ventral direction, so that on the transverse section it appears as a long fissure directed dorso-ventrally.

The morphological transformations of the gray matter occurring in the conus terminalis need not be dwelt upon. Another point, however, needs mentioning. No actual lateral-horn formation is found in the sacral region of the cord. The marked projection which is seen at certain levels, in a position corresponding to the lateral horn, is not homologous to the lateral horn of the dorsal levels; for it is occupied by a group of large cells of the characteristic anterior-horn type, while the lateral horn of the dorsal levels contains a group of small cells known as the lateral-horn group or tractus intermedio-lateralis. But although an actual lateral-horn formation is absent in the sacral portion of the cord, this region contains nevertheless a group homologous to the lateral-horn group or tractus intermedio-lateralis of the dorsal region. (But of this later.) In concluding, it is interesting to remark that owing to the morphological transformation mentioned, the configuration of the gray matter of the first sacral segment resembles much more

that of the fifth lumbar than that of the second and other sacral segments. This relationship, in considerable degree, holds true also for the cell groups of this region, and one comes to the conclusion that structurally the first sacral segment belongs

ANTERIOR-HORN GROUPS OF THE LUMBAR AND SACRAL REGIONS OF THE HUMAN SPINAL CORD.

	Mesial		Antero-lateral Group I		Postero-lateral	Post-postero-lateral	Central and Middle	
	Antero-mesial	Postero-mesial	Antero-lateral	Group I			Centr.	Middle
I. Lumb. segm.	●	•	•		•		•	
II. Lumb. segm.	●		•		•			•
III. Lumb. segm.	•		•					•
IV. Lumb. segm.	•		•		•		•	
V. Lumb. segm.	•		•		•		•	
I. Sacr. segm.		•	•		•	•	•	
II. Sacr. segm. } prox. part } dist. part }	• • •		• • •	• • •	• • •	• • •	• • •	
III. Sacr. segm. } prox. part } dist. part }	• • •	• • •	• • •	• • •	• • •	• • •	• • •	
IV. Sacr. segm. } prox. part } dist. part }	• • •	• • •	• • •	• • •	• • •	• • •	• • •	
V. Sacr. segm.								

Fig. 1. The above table represents the distribution and relative size of the anterior-horn groups of nerve cells in the various segments of the lumbar and sacral regions. The sizes given are not based on a count of the nerve cells, but only on a rough estimate from serial study.

more properly to the lumbar than to the sacral portion of the spinal cord.

Arrangement of the cell groups of the sacral region :

The cell groups of the sacral region may be conveniently classified into (1) Anterior-horn groups and (2) groups or cells which are not of the anterior-horn type.

1. The anterior-horn groups.

The adjoining diagram gives an idea of the anterior-horn groups found in the sacral region, showing approximately their relative size at the various sacral levels and their extent in cephalo-caudal direction.

Some explanatory remarks may be desirable regarding the post-postero-lateral group. At the level of the first sacral segment three lateral anterior-horn groups are found: the antero-lateral, the postero-lateral and the post-postero-lateral. At the level of the second sacral segment the antero-lateral group disappears and the group "X" arises in its place, moving slightly mesiad. The postero-lateral group grows smaller, moves forward and becomes *de facto* antero-lateral. The post-postero-lateral group becomes situated dorsad of the postero-lateral group, thus being actually postero-lateral in its position.

While the antero-lateral, the postero-lateral and the post-postero-lateral groups consist of large multipolar cells, the elements constituting the group "X," although also multipolar, are characterized by their relatively small size and their rather densely packed arrangement.

2. Groups or cells which are not of the anterior-horn type.

(a) Scattered large cells at or near the base of the posterior horn.

(b) A cell formation which I propose to call the vegetative cell column (See Fig. II), and which can be subdivided into a lateral and central division. The lateral division may often be divided into a dorsal and a ventral part. The lateral division of the vegetative cell column corresponds apparently to the tractus intermedio-lateralis. The central division is probably identical with the paracentral group which Dr. Joseph Collins and I described in a monograph on the representation of the sympathetic nerve in the spinal cord and medulla oblongata, which will be published in the *Archives of Neurology and Psychopathology*, and an abstract of which has appeared in the JOURNAL OF NERVOUS AND MENTAL DISEASE, 1898, p. 661.

The arrangement of the cell groups is so different in the various segments of the sacral portion of the cord that study of a number of consecutive transverse sections will reveal rather exactly the level or segment from which they are taken, especially when the gross configuration of the gray matter is taken also into consideration.

The first sacral segment, as compared with those below, is characterized by the shape of the gray matter, which resembles much more that of the fifth lumbar than that of the second and other sacral segments, especially with reference to the small

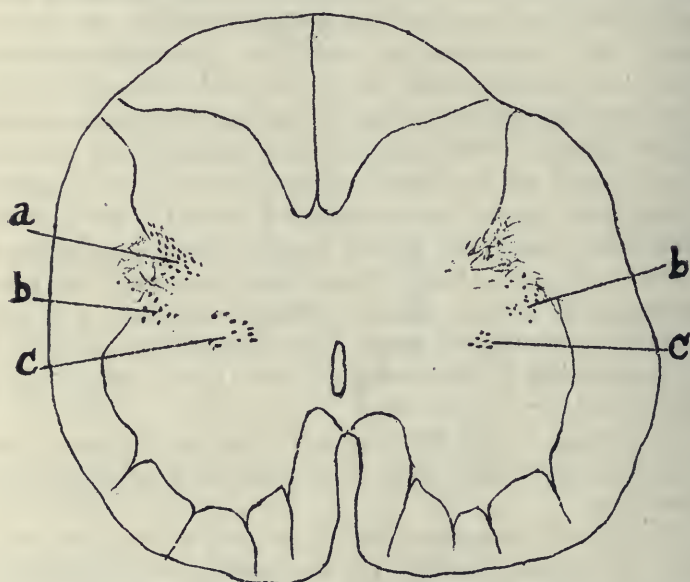


Fig. 11. Third sacral segment, showing the vegetative cell column (veget.) in its three divisions: the dorso-lateral (a), the ventro-lateral (b) and the central (c).

depth (dorso-ventrad) of the gray commissure and the circular or only slightly oval shape of the central canal; furthermore, by the presence of the three lateral groups (antero-lateral, postero-lateral and post-postero-lateral), although the post-postero-lateral group is represented by few cells only. There is further almost complete absence of a mesial cell group.

The second sacral segment is characterized by the presence

of the group "X" which is almost typical for this segment, although it extends slightly also into the distal part of the first and the proximal part of the third sacral. The distal part of the second sacral segment is recognized by the appearance of an antero-mesial group which is absent in the proximal part.

The third sacral segment shows off by the presence of two mesial groups, namely, an antero-mesial and a postero-mesial, and by the marked and typical development of the vegetative cell column, especially of its central division.

In the fourth sacral segment the anterior-horn groups are practically gone, the mesial ones, and especially the postero-mesial group being the last one found. The anterior horn is beset with a great quantity of small cells. The lateral division of the vegetative cell column is still rather well developed, contains a considerable number of larger cells, and shows a tendency of extending all along the lateral border of the neck and head of the posterior horn.

In the fifth sacral segment it is chiefly the shape and size of the gray matter that distinguishes it from the fourth sacral, the anterior horn having become very short. Still further caudad the gray matter changes its aspect altogether.

Suggestions regarding the functions of the cell groups in the sacral region of the spinal cord.

I give here only the conclusions arrived at:

(1) Function of the antero-lateral, postero-lateral and post-postero-lateral groups: These three groups are evidently presiding over the muscles of the lower extremities, to judge from analogy with the cervical region, and from the researches of Van Gehuchten and de Buck,³ and Sano.⁴

(2) The antero-mesial groups is evidently the continuation of Kaiser's⁵ nucleus of the back muscles which, he finds, extends as a mesial column throughout the length of the spinal cord.

(3) The postero-mesial group is probably the nucleus for the perineal muscles, and probably the center for the spincters ani et vesicæ forms part of this nucleus.

(4) The group "X" possibly, or even probably, is a center

⁵Kaiser, O.: "Die Funktionen der Ganglienzellen des Halsmarkes." Prize essay, Hague, 1891.

for some of the striated muscles which co-operate in the acts of erection and ejaculation, especially the ischio-cavernosus or the erector clitoridis, and the bulbo-cavernosus or the sphincter vaginæ muscles.

(5) The cell column "veget" or vegetative cell column, in conformity with its strong development in the third sacral segment, is evidently a motor and vasomotor representative of important vegetative functions located in this and the fourth sacral segment, to the extent to which these functions are performed by means of non-striate muscles, namely: the function of emptying the bladder (detrusor vesicæ muscle), that of erection and ejaculation, of emptying the rectum and (in case of pregnancy for instance) probably also of emptying the uterus.

Müller's article appeared after my researches had already been completed and their full report sent in for publications.

151. UEBER "SPRINGENDE PUPILLEN" IN EINEM FALLE VON CEREBRALER KINDERLÄHMUNG, NEBST EINIGEN BEMERKUNGEN ÜBER DIE PROGNOTISCHE BEDEUTUNG DER "SPRINGEN DEN PUPILLEN" BEI NORMALER LICHTREACTION (Concerning "springende Pupillen" in a Case of Infantile Cerebral Paralysis with Some Remarks on the Prognostic Value of "springende Pupillen" Associated with Normal Light Reaction). W. Koenig (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, Nos. 1 and 2, p. 122).

By "springende Pupillen" is meant a condition in which mydriasis is seen first in one eye, then in the other, so that the relative width of the two pupils varies frequently. Koenig has observed this condition in a case of infantile cerebral paralysis, the first case of the kind on record. This variation in the width of the pupil has been regarded as an ill omen; Koenig thinks:

1. It is rare, but occurs with pupils reacting normally as well as pathologically.

2. It has been observed chiefly in organic diseases of the central nervous system, more rarely in functional diseases of this system, and very seldom in perfect health.

3. It is of prognostic value only when the reaction of the pupil is normal.

4. It is not necessarily a bad omen when the pupillary reaction is normal and no signs of organic nervous disease are present. It may, however, be an early sign of paretic dementia.

5. Differences in the intensity of reaction of the pupils, especially unilateral rigidity of the pupil, may lead to a mistaken diagnosis of "springende Pupillen."

SPILLER.

PHILADELPHIA NEUROLOGICAL SOCIETY.

April 24, 1899.

The President, Dr. James Hendrie Lloyd, in the chair.

A CASE OF FRIEDREICH'S ATAXIA.

Dr. Elizabeth L. Peck presented a girl of seventeen years with this disease. The father was healthy. She had two brothers, one older and one younger than herself, both well. The mother suffered from the same trouble as the patient, and was helpless two years before death, which occurred eight years ago. Mother's sister also suffered from the same affection.

The patient thought she had always had some difficulty in walking, though she remembered that she could run and jump rope, and that she went to school until after she was fourteen, and stood fairly in her classes. She had great difficulty in learning to write. Had never had any serious illness. For the last two years she had had great difficulty in walking and in holding articles. Her gait was remarkably ataxic, and movements of her head and body were seen even while she was sitting. The tongue was tremulous. No changes in the pupillary reactions and no nystagmus were noticed. The speech was rather slow. The knee-jerk and tendo-Achillis jerk were absent, and the foot showed the hollow instep and hyperextension of the toes characteristic of the disease. No spinal curvature, no lancinating pains, no involvement of the sphincters, and no disturbance of sensibility were present in this case.

Dr. H. F. Hansell and Dr. W. G. Spiller presented a tabetic patient with bilateral ptosis. Bilateral ptosis is not of very common occurrence in tabes, although it is well known. This was one of two cases of tabes with bilateral ptosis at that time in the Polyclinic Hospital.

Dr. C. K. Mills presented a case of anomia.

A CASE OF MERCURIAL POISONING.

Dr. J. K. Mitchell presented a man of German origin, born in Philadelphia, aged 36, watchcase engraver, who was admitted to the Orthopedic Hospital, March 7, 1897, with the following history:

In the month of October, 1896, the patient, suspecting that he had pediculosis, used a vigorous inunction of mercurial

ointment, rubbing in two ounces of the salve. The next day, not satisfied with this, he used two ounces more. Within twenty-four hours he was seized with violent intestinal pain and furious diarrhea, followed by vomiting. The digestive disturbance continued for some days. In the course of the succeeding five weeks he lost about fifty pounds in weight. Numbness in the hands and feet began about two weeks after the inunction, and was soon followed by violent pain in the forearms and lower limbs of shooting, stabbing, lightning-like character, with only the shortest interval between. The patient said that for many weeks he was scarcely ever ten seconds without pain. Pain was still great when he was admitted to the hospital. There was at no time any affection of the mouth or gums. About three weeks after the inunction he noticed some weakness in the hands, and very soon afterwards in the lower legs. This increased until there was complete paralysis of all four extremities, with wrist-drop and ankle-drop. In this condition he remained for about three months before he was admitted to the Orthopedic Hospital.

When first seen there his forearms were much wasted and the fingers a little contracted, and the whole hand very much atrophied. He was unable to oppose the thumb to the little finger, though he could bend it part way, and his grasp was exceedingly feeble. There was slight tenderness over the nerve trunks in the forearms, not extending much above the elbow. A similar condition was present in the legs. He could not flex the foot at all upon the leg, and was wholly unable to stand, the muscles up to the knee sharing in the atrophy.

When he related the above history he was closely questioned as to the possibility of any other form of toxic influence than that of mercury. His work occupied him only with the engraving of gold watch cases, and he was a man of excellent character and habits, never given to constant or excessive use of alcohol, or to any other form of undue indulgence. His hair was falling rapidly when admitted and he lost a great portion of it, though some of it came back afterwards. At the same time there developed a mark upon the upper part of the forehead running diagonally from the middle of one temple to the upper part of the other, and including the forehead down to the eyebrows in a faint, but decided brownish stain, like chloasma, which was very conspicuous upon his fair skin.

His improvement was very slow, but quite constant, under daily massage, faradism to the enfeebled muscles, and later, alternating applications of hot and cold water to the extremities.

He has completely recovered, with the exception of slight weakness in the anterior tibial muscles, rather more marked

on the left side than on the right. The strength of the arms is perfect in all muscles, and his control of the hands good enough for him to take up massage as a profession, from the interest which he acquired in it at the hospital, and he is doing excellent daily work in that line.

The interest of the case lies in the mode of infection, in the absence of the classical stomatitis of mercurial poisoning, and in the completeness of his recovery from the toxic neuritis.

Dr. F. X. Dercum said that it was remarkable that there had been no salivation. This was contrary to all experience with mercurial poisoning by inunction. The pigmentation was also an unusual feature. Pigmentation is noted in poisoning by other metals, especially in the case of arsenic, but in the latter instance it is not as evenly diffused or as sharply delimited.

A CASE OF TETANY.

Dr. Elizabeth R. Bundy presented an Italian woman, 32 years of age, who had three children and had been ten years in America. Seven years ago the first attack of tetany occurred while she was nursing her first child. Several attacks have occurred since, light in character, always in the spring, and more noticeable during lactation. The spasms were always confined to one or both upper extremities. The present attack had been of three months' duration, and was the most severe of all the attacks. Trousseau's, Chvostek's, Erb's and Hoffmann's sign were present. The case was a typical one. This was the second case of tetany from the Polyclinic Hospital that Dr. Bundy had presented before the society within a short time.

The President remarked that three years ago he had reported to the society a case of marked tetany in an Italian who had been in this country two years. The symptoms of tetany were exaggerated and Trousseau's sign was brought on by pressure on the nerve trunks of the arm. There was distinct spasm of the facial muscles on tapping the facial nerve. The man died not long afterwards with obscure febrile symptoms, which did not seem to have any connection with tetany.

A CASE OF HYSTERICAL TREMOR.

Dr. Charles W. Burr presented a patient with hysterical tremor. She was a single woman, about 30 years old. The family and personal histories were negative. When about 13 years old she began to complain of queer feelings in the back, like water trickling under the skin, coming on only during emotional excitement. Three years later the right arm began to twitch, two years after that the head, and finally the entire body became involved. In 1894 an operation was per-

formed upon both sides of the neck. Judging from the position of the scars, it is probable that the spinal accessory nerves were cut. The operation was not followed by any relief.

She was a fairly well nourished, healthy-looking woman. When she was alone or with people whom she knew well she did not suffer in any way or present any evidence of the remarkable tremor which developed under the least excitement. She could do kitchen work without difficulty, but if the bell rang, or if she was suddenly called, or some one approached unexpectedly, she immediately began to shake from head to foot. The movements were jerking and violent. The endeavor to overcome them while walking gave her a curious, stiff gait. At times one leg flew out and she fell to the floor. Speech was jerky and tremulous. She presented no other evidence of disease. Sensibility was normal to all forms of stimuli. The deep and superficial reflexes were normal. The ocular examination made by Dr. Charles A. Oliver showed nothing abnormal. Muscular strength was good, and there were no palsies. The urine did not contain albumin, casts, or sugar. The thoracic and abdominal organs were normal. Intelligence was fair, and the patient presented no other sign of emotionalism save that excitement caused the attacks of tremor.

Though she presented no other stigma of hysteria, Dr. Burr was forced to the diagnosis of hysteria by exclusion. She had none of the other symptoms that accompany the other diseases in which violent jerking movements occur. The one point against hysteria was the entire absence of other stigmata, but monosymptomatic hysteria does occur, and it is possible that if we knew her entire history we would find that she had had at some time anesthesia, or palsy, or convulsive attacks.

Dr. J. W. McConnell said that he recalled the case of a young woman, a private patient of Dr. Mills, in which there was general tremor more marked in the lower extremities, and coming on under similar circumstances. She recovered perfectly. He had since seen her several times riding in the park on a bicycle.

Drs. Musser and Sailer reported five cases, three of infectious disease and two of anemia, that presented symptoms or lesions in the nervous system. The first, a girl of 15, had increased reflexes, ankle-clonus, intention tremor, and nystagmus. At the autopsy the lesions of acute septicemia and infectious nephritis were found. The nervous system was not examined. The second and third patients had typhoid fever; both had exaggerated tendon reflexes and ankle-clonus. A slight tremor was present. One recovered, and developed loss of knee-jerks and Romberg's symptom; the other patient died, and changes were found in the spinal cord. The fourth pa-

tient was suffering from severe anemia, with marked reduction in the hemoglobin. The tendon reflexes were greatly exaggerated. There were ankle-clonus and slight Romberg's symptom, but no ataxia. The last patient had had profound anemia of eight years' duration, following smallpox. Death occurred as a result of erysipelas. At the autopsy small myelitic foci composed of a central mass of necrosis with round cell infiltration were found in the gray substance of the spinal cord.

Dr. J. W. McConnell read a paper with the title: "Transient Paralysis as an Epileptic Equivalent." (See p. 355.)

A CASE OF PARALYSIS LIMITED TO THE UPPER EXTREMITIES.

Dr. A. Ferree Witmer reported the case of a colored man, 28 years of age, who two months previous to the examination had noticed weakness in the right hand, with inability to fully flex the fingers of this hand. Wasting in the right arm was observed two weeks later. One month after the apparent onset of the trouble the patient complained of severe pain in the right side of the neck, radiating down the right arm. The severity of the pain at that time prevented sleep. Three weeks later wasting began in the left arm. No pain was ever felt in the left arm.

Examination showed the head inclined forward and to the left. Pupils were regular and active for light, and in accommodation. The patient could shrug the shoulders on both sides, but the pectoral muscles on both sides were very weak; the trapezius stood out slightly, possibly also the rhomboids. Decided wasting was noticed in the suprascapular region; less in the infrascapular region.

Right upper extremity: The deltoid was very weak and greatly atrophied, and the patient had no power of elevation of the arm. The flexion at elbow was only at an angle of about 120 degrees. The triceps was paralyzed, and supination and pronation of the forearm were abolished. Some power of extension and flexion at the wrist was preserved, but little of extension in the fingers. Flexion and opposition of the fingers were practically nil.

Left upper extremity: Paresis differed only in not being so marked as in right extremity.

The electrical examination of the upper limbs with the continued current showed no serial changes, although responses were not so prompt as normally. On both sides the flexor groups were more active than the extensors. The interrupted current caused some contraction of all the muscles, excepting

the extensor communis digitorum. No tenderness was observed on free manipulation of the arms. The sensation to touch was normal. The knee-jerks were much diminished, but quite perceptible on reinforcement. No ankle-clonus and no weakness of the lower limbs were noted.

The symptom-complex in this patient was so extraordinary that the affection could not be classified under any of the ordinary types, but it was thought by Dr. Witmer to be an acquired neuronal degeneration of the motor type, with an anomalous condition of the knee-jerk, unless it were assumed that in this patient the knee-jerks were normally greatly diminished.

Dr. William G. Spiller said that the case had been sent to the clinic for nervous diseases at the Polyclinic Hospital, by Dr. S. Solis Cohen. It might properly be called one of brachial paraplegia. It was interesting to note the definition of paraplegia as given by some dictionaries. Isolated paralysis of the lower limbs is so much more common than isolated paralysis of the upper that the word paraplegia conveys to the minds of many the thought of paralysis in the lower limbs. This case presented almost complete paraplegia of the upper limbs, without any weakness of the lower limbs. When the arm was laid on a table the patient could not extend, supinate or pronate it.

The symptoms began with weakness in the right upper limb, followed by intense pain, and after a few weeks by weakness in the left upper limb. Dr. Spiller thought that the case was one of cervical pachymeningitis, or possibly multiple neuritis. Involvement of the spinal cord could probably be excluded, and the disease was evidently one of the peripheral neurons.

Dr. Spiller also exhibited some slides illustrative of spinal pachymeningitis.

Periscope.

ANATOMY AND PHYSIOLOGY.

152. NERVENZELLEN UND GRAUE SUBSTANZ (Nerve Cells and Gray Matter). Nissl (Münchener medicinische Wochenschrift, 1898, Nos. 31, 32, and 33.)

Alluding to his most recent experiments on the action of various poisons upon the nerve cells, and pointing out the various sources of error, the author takes as his starting point a disease of the cells of the human cortex, which he says he has best studied, and which he calls the "acute cell disease." In this all the cells of the cortex are affected, the whole cell body suffering, and the changes in them are entirely characteristic. These changes show that the unstained part of the cell consists not only of fibrillary substance, but also of something else. This acute cell disease is found in acute cases of paresis, in various psychoses, and also in patients dead of other diseases, who prior to death have been delirious or somnolent.

Recent investigations all seem to show that the functionally active part of the cell is that left unstained in Nissl's method, and through the masterly researches of Apathy, and of Bethe, this has acquired the greatest importance. "The quintessence of the newer investigations consists in the fact that the nervous tissue can no longer be considered as a community of countless nerve cell individuals, which possess, so to say, some a greater, others a smaller, radius of action, but we must regard it as composed of nerve cells, and a specific nervous substance, namely, the fibrillary substance."

This is a living substance, a specially differentiated nerve cell protoplasm in its highest development, and appears to be directly concerned in carrying on nervous functions. The conducting element is undoubtedly the primitive fibrils. The neuron conception, the author thinks, is hardly reconcilable with the recent advances in knowledge of the histopathology of the nervous system, since it was originally based upon the Golgi method, and we do not know to-day just to what tissues the blackening by this is limited. He next proceeds to discuss the gray matter in its histological constitution, and points out that the usual view, that it is a part of the nervous system, in which nerve cells and neuroglia cells and fibers predominate, does not explain all its peculiarities, since in some parts of the gray matter cells are very few in number, while again, the white matter in certain regions contains a good many cells. The criterion for the distinction between gray and white matter he finds not in the relative abundance of nerve cells and nerve fibers, but in the presence or absence between the cells, fibers and vessels, of a peculiar molecular substance which gives the gray color. This substance, he thinks, must be regarded as an active factor in the mechanism of the nervous system, and probably is made up largely of the fibrillary substance already mentioned, though positive proof of this has not yet been secured. Certain it is, that in the functionally highest parts of the nervous system there is, along with the nerve cells, something which is made up neither of neuroglia nor of the sum of the processes of nerve cells. To show this the author gives pictures of sections taken from as near as possible corresponding portions of the

cortex in the posterior part of the motor region, in man, the dog and the mole. In these it appears that in the second and third Meynert's layers there is a difference in the proportion of cells and intercellular substance, the former being less, the latter being more abundant, the higher in the scale of intelligence we rise. The author has always claimed, in opposition to many authorities, that there is a difference in structure in the nerve cells from functionally different portions of the nervous system. The specific nervous substance is in very close relationship with the nerve cell. It is found, first, in the cell body—and here, according to Bethe, there are to be distinguished central fibrils and peripheral fibrils; second, it surrounds the cells with their dendrites as “a true closed and rather close-meshed net.” (Held.) The dendrites seem to be designed for bringing cells into extensive relations with the specific nervous substance, and, considering the arrangement of the axis cylinder processes, the nerve cells seem to be “collection stations” for the fibrillary substance, “in which the fibrils undergo a rearrangement in behalf of conduction into other processes, and forming the axis cylinder process.” The axis cylinders, coming together, form important nerve tracts. It must not be thought, however, that the nerve cell plays but a subordinate rôle in the work of the central organ, for while the researches of Bethe show that in invertebrates reflexes can be excited without the intervention of a cell these reflexes are feeble, and are soon exhausted.

In no case of psychical disturbance has the author failed to find a number of the cortical nerve cells diseased. The nerve cell is sterile, as far as reproducing other nerve cells is concerned, but is able to reproduce the fibrillary substance in large quantity. How is not known. The author next considers the portion of the cell staining with methylene blue, the tigroid or Nissl bodies. This is improperly called, since it is not homogeneous, but is composed of several substances which react differently in different cells. The nucleus next engages his attention; he shows that its structure is complicated and varies in different cells, and that it is composed of several differently reacting substances. The increase of capability, as we advance from the lower to the higher order of animals, is due to the ever greater division of labor among the individual cells, through their greater specialization. By the nerve cells, the nutritive functions of the gray matter and fibril tracts are regulated, and a condition of tension ready to set at liberty nervous impulses under the slightest irritation is produced. The position of the nerve cell as a collecting station has already been alluded to, and the question is raised if there are not fibrils which, running in the collaterals, enter the main axis cylinder without touching the nerve cell, a condition which Bethe has shown to exist in the case of the dendritic fibrils. However, even assuming this to be the case, the nerve cell does not lose its importance, since its intimate connection with the specific nervous substance cannot be denied. Both arise from the same basis, and their separation into nerve cell and nerve substance is interesting chiefly as an example of division of labor.

ALLEN.

153. APUNTES PARA EL ESTUDIO ESTRUCTURAL DE LA CORTEZA VISUAL DEL CEREBRO HUMANO (Observations on the Structure of the Visual Cortical Area in Man). S. Ramon y Cajal (*Revista Ibero-Americana de Ciencias medicas*, March, 1899).

Cajal has given an extensive study with a complete literature on the structure of the cuneus and the cortex bordering on the calcarine fissure. This is but one of the author's many morphological studies on the cortex of the human brain. From without inward in the region of the cuneus he distinguishes eight different zones, as follows: 1. The

molecular layer. 2. The layer of small pyramids. 3. Layer of medium sized pyramids. 4. Stellate layer or layer of Gennari. 5. Layer of small spheroidal cells. 6. Layer of giant pyramids. 7. Layer of polymorphous cells. 8. White substance. These layers are again divided, but the original should be consulted. In brief, the outer three layers are more or less typical. The layer of Gennari presents three structures: large stellate cells, large ascending fibers, which, by their rich ramifications, make up the horizontal striations of this layer, and axonal and collateral ascending fibers from the stratum below.

The layer of polymorphous cells consists of at least four types of cells.

JELLIFFE.

PATHOLOGY.

154. ZUR HISTOLOGIE DER POLIOMYELITIS ANTERIOR CHRONICA (Histology of Chronic Anterior Poliomyelitis). Max Bielschowsky (*Zeitschrift für klin. Med.*, Vol. xxxvii, Nos. 1 and 2, p. 1).

The patient described in this paper was seventeen years old. He was said to be as healthy as normal boys until his ninth year. At that age, without known cause, a flaccid atrophic paralysis began, and later involved the entire musculature of the extremities and trunk. The paralysis was first noticed in the lower limbs in the peroneal group. In the later stages of the disease the muscles of the neck were affected and the tongue was atrophied and paretic. The paralysis in the beginning of the disease preceded the atrophy; later this mode of involvement was not so noticeable, but the paralysis was always more pronounced than the atrophy. Contracture and kyphoscoliosis developed in the course of the disease. The diagnosis of chronic poliomyelitis was made. The contractures, which were of an extreme type, were not spastic but paralytic, and developed later than the atrophic paralysis. The tendon reflexes were absent and the paralyzed muscles were flaccid. The motor cells presented marked quantitative and qualitative changes throughout the cord as high as the hypoglossal nucleus, and the anterior horn cells which are nourished exclusively by the central artery, were most diseased, while those near the periphery of the anterior horn, which receive some nourishment from the peripheral arteries, were not quite as much diseased. Cells other than motor in the anterior horns were also affected. The nerve fibers and neuroglia fibers of the anterior horns were diminished in number; the intramedullary portions of the anterior roots were much degenerated; the vessels of the anterior horns were diseased and new vessels were formed; round cell infiltration, recent and old hemorrhages were seen in the anterior horns. The lateral pyramidal tract showed no degeneration except by the Marchi method, and even by this method the degeneration was insignificant.

SPILLER.

155. STUDIES ON GANGLION CELLS. J. Ewing (*Archives of Neurology and Psycho-pathology*, I, 1898, p. 283).

Ewing here gives, in a monograph of 180 pages with six plates, the most comprehensive contribution to ganglion cell pathology yet published. For wealth of material, character of research, bibliographical completeness, excellence of typography and high class illustrations, this monograph is exemplary.

He first discusses the subject of technic and gives a full bibliographical summary and advises the use of alcohol, 97 per cent.; alcohol and corrosive sublimate; formalin sublimate; Van Gehuchten fluid and formalin, 10 per cent, as of about equal service, especially when small pieces of tissue are used. The author's views with reference to

the histology of the cell is in close accord with those of Dogiel and Held, but his interpretations are wider than those of most authors. The physiological conditions and the general variations in structure of the ganglion cells in cases dying of hemorrhage and shock, and cadaveric changes in ganglion cells are fully discussed. With reference to the last the opinion is expressed that there is no evidence to show that cadaveric changes can simulate those produced by disease processes. Cell changes in diseases of the nervous system are then taken up, the observations of recent authors being corroborated and amplified. Ewing does not believe that it has been demonstrated that primary and secondary affections of the neuron in the neuritic process can be clearly differentiated by any method at our command at present. The literature on myelitis, Landry's paralysis, tabes, bulbar palsy, paresis and the insanities and idiocy are reviewed, but no original observations are recorded. Investigation of two cases of carbolic poisoning showed an absence of clearly defined lesions, notwithstanding grave nervous symptoms, and in three cases of eclampsia few changes were noted. Six cases of uremia showed marked alterations in the chromatic structures, irregular in character and distribution. The medullary nuclei, notably the tenth nucleus, were most involved. Three cases of sunstroke and numerous cases of hyperpyrexia in rabbits lead the author to believe that in this condition the alterations are always pathognomic in character. In various forms of meningitis the cell changes seemed most marked in the compressed and anemic areas, and led the author to infer that grave circulatory disturbances produce more cellular modifications than soluble toxic materials. In two cases of typhoid, marked cellular lesions distributed throughout the nervous system were observed, probably influenced largely by the hyperpyrexia. Acute lobar pneumonia may or may not induce cellular lesions; all the cases here recorded, three in number, showed some involvement of the medullary nuclei. In cases of general sepsis from bronchitis, pyemia, empyema, phlegmonous inflammation, and tubercular infection, wide spread in character, chromatic alterations were observed in each case. The severity of the cellular lesions seemed in proportion to the amount of toxemia and the temperature. Tetanus and hydrophobia showed changes similar to those described by other writers. Ewing then discusses the general significance of the Nissl bodies and the bearing of them on nutrition and allied conditions making a complete summary of the recent literature. He also reproduces a number of the experiments of Goldscheider and Flatau on hyperthermia, and his results in the main agree with those of these investigators. The writer's general conclusions of the general characters of the lesions in nerve cells is worthy of quotation *in extenso*, but space compels abstracting. He believes that in the general process of chromatolysis the first change is that of a preliminary swelling of the chromatic body, best shown in the cases of tetanus and hyperpyrexia in the early stages. A uniform diminution then results, to be followed by a uniform subdivision of the chromatic bodies, such changes being observed in the chronic toxemias. Granular subdivision of the chromatic bodies may result as a further stage from sudden acute lesions. The final stage in the process, in which the cell body has lost all of its stainable substance, is the "simple complete chromatolysis." This result seems to follow a variety of causes and no hint as to the origin can be gained from the pathological picture. Minute variations in the chromatolytic process appear in nearly every case and any attempt to exhaust all of the possible peculiarities is held to be futile. "The minute lesions appear to differ in every case and the range of minor peculiarities is practically limitless," are the words of the author. When acute degeneration

eration, in a strict sense, affects the ganglion cell, its changes are evidently manifested by various forms of chromatolysis, but the true degenerative process may not, and as the writer believes, "frequently does not begin until after chromatolysis is complete." JELLIFFE.

156. LA TOXICITÉ DE LA SUEUR CHEZ LES ÉPILEPTIQUES ET LES MÉLANCOLIQUES (Toxicity of the Sweat of Epileptics and in Melancholia). Dr. Mavrogiannis (*Revue de Psychiatrie*, 2, 1898, p. 199.)

Cabitto's researches on the sweat in epileptics were so startling, not to say apocryphal, that it is not surprising to find that his conclusions have been called in question, and, according to the author's experiments, disproven. In a series of experimental injections in rabbits of post-paroxysmal perspiration the results were almost negative. In a few cases there were muscular reactions, but these could be explained on other grounds. The secretions from cases suffering from melancholia were also nontoxic. [A few more such conscientious experiments would probably modify the Bouchard methods, in which connection the recent work of Putnam-Pfaff is to be consulted as a model of careful research.] JELLIFFE.

157. ANCORA SULLA GLIOSI CEREBRALE NEGLI EPILETTICA (Cerebral Gliosis in Epilepsy). A. Tedeschi (*Policlinico*, 6, 1899, No. 1).

In a recent case of epilepsy the author describes a marked asymmetry of the two hemispheres. The left side was smaller than the right and showed a peculiar structure. All of the structural elements were much simplified, but there was a marked increase in the neuropilgia, making a distinct felt-like tissue with few nerve cells and many fibers. There were no alterations in the vessels nor in the meninges. The nerve cells of the right hemisphere were normal, as were the ganglion cells of the basal ganglia, though the left half had fewer elements. There were fewer Purkinje cells in the left half of the cerebellum. JELLIFFE.

158. OBDUCTIONSBEFUNDE BEIM TOD IM STATUS EPILEPTICUS (Autopsy findings in Status Epilepticus). L. W. Weber (*Wien. med. Wochenschrift*, 1899, p. 157).

Weber has given one of the best microscopical studies of the nervous system in severe epilepsy that has been contributed of recent years. His results were drawn from a series of autopsies with microscopical examinations carried on for the past four years in the asylum of Uchtspringe. The most constant lesions were found in the blood vessels. These in the brain and cord and medulla were found to be filled with blood and markedly dilated. In places there were blood infarctions with hyaline degeneration of the walls of the capillaries. In many cases there were extravasations of red blood cells into the lymph spaces and perigangliar spaces. In many of these extravasated areas the blood cells had undergone retrograde changes, and in some instances destructive lesions of the ganglion cells were observed. The ganglion cells of the spinal cord were often found to be rich in pigment, and the vascular lesions were of the same type. The author holds that these vascular degenerations are secondary rather than primary, and looks for the primary cause in some form of endogenous poisoning. The transitory paralyses and pareses, he thinks, are to be attributed to these minute lesions, but the epileptic attack itself is the result of some form of auto-intoxication. When the lesions involve the cranial nerves death may result. JELLIFFE.

CLINICAL NEUROLOGY AND PSYCHIATRY.

159. UEBER EINE NACH ETIOLOGIE, KLINISCHER VERLAUF, UND PROGNOSE, GENAU ABGRENZBAREN, SICH ALS ALCOHOLOGENE, CARDIALE EPILEPSIE CHARACTERISIRENDE GRUPPE EPILEPTOIDER ZUSTÄNDE (On a Group of Epileptoid Conditions, Characterizing Themselves as Alcoholic Cardiac Epilepsy). Smith (Münchener medicin. Wochenschrift, 1898, No. 43, p. 1372).

By the use of the phonendoscope, and of the Röntgen photography, the author claims to be able to demonstrate an increase in size of the heart in every case of chronic alcoholism, and states that these patients are particularly liable to epileptiform attacks, presenting themselves under two chief groups. In the first, without any warning, the patient suddenly passes into a sort of delirium (dämmerzustand) which lasts several days, and on recovery from which there is no recollection at all of what has occurred.

In other cases the patient experiences—most commonly on rising in the morning—a feeling of discomfort, soon becoming one of distress, with pain over the cardiac area, in many instances followed at once by epileptiform convulsions. The sufferer is often impelled to seek relief in alcohol, and may obtain temporary ease, but he soon passes into a condition of delirium, often accompanied by violent acts, of which there is no recollection when the attack is over. In both classes of cases there is always an acute enlargement of the heart, which under abstinence and proper treatment may disappear and the patient remain well. In the second class of cases—usually in those having a strong nervous heredity—while the enlargement of the heart has diminished and the patient is improving, about the fourth to sixth week he is noticed to be getting restless and irritable, especially in the morning, the heart is found to be again enlarged, the pulse is quickened (100 to 140), and the patient complains of pain over the cardiac area. He becomes suspicious and depressed, and is upset by the slightest thing. Should he seek the aid of alcohol, he will very likely have an epileptiform attack.

If alcohol can be rigidly withheld, however, and the intervals between the attacks be utilized for proper tonic treatment, the heart may become normal and remain so. The author further discusses the diagnosis and treatment. The best results were obtained from trional and prolonged baths during the attacks, with general tonic measures, hydrotherapy, Swedish movements, and especially outdoor exercise, in the intervals.

ALLEN.

160. UN CAS D'ASSOCIATION DU TABES ET DU GOITRE EXOPHTHALMIQUE (A Case of Association of Tabes with Exophthalmic Goiter). Delearde (Gazette hebdomadaire de med. et de chir., Jan. 15, 1899).

The simultaneous appearance of these two diseases in the person of the same subject is no novelty, but the author wishes to consider the question as to whether the conjunction of the two maladies is purely fortuitous or not. There are two reasons for a difference of opinion in this respect. In the first place, the relative dates of appearance of the two, and in the second place the absence of lesions of the nervous system characteristic of goiter. In the case under discussion the goiter preceded the tabes by ten years, but was subsequent to an attack of syphilis.

Is the Basedow's disease to be considered as secondary to the syphilitic infection and induced by changes produced by this in the thyroid? Or, granting that the gland escaped affection by the luetic virus, may not the nervous system have been predisposed by it to the goiter, regarding the latter purely as a neurosis? Whatever conclusion is reached, it is evident that in this case the goiter was not the result of the extension of tabetic lesions from the cord to the bulb.

JELLIFFE.

161. EIN FALL UNCOMPLICIRTER SERRATUSLÄHMUNG NACH INFLUENZA (A Case of Uncomplicated Serratus Paralysis after Influenza). Von Rad (Münchener med. Wochenschrift, 1898, No. 36, p. 1145).

Uncomplicated paralysis of the serratus magnus muscle is a rare condition, hence the author has put on record a carefully prepared description of a case of this disease, coming on after influenza in a man of thirty-four, of good family and personal history. The article is illustrated by two reproductions of photographs. ALLEN.

162. TABES DORSALIS UND SYPHILIS. A. Guttman (Zeitschr. f. klin. Med. 35, 1898, p. 242).

The reaction, evident of recent years, against the view of the etiological significance of syphilis in tabes, is made more pronounced in the statistics of the author. In all some 136 cases of tabes were closely studied, and of these, deducting six doubtful cases, 28.6 per cent. had had syphilis, while 71.4 per cent. were distinctly nonsyphilitic. The author also considers the evidence derived from the fruitlessness of syphilitic medication, and gives his approval of the general methods used to strengthen the body—baths, massage, and electricity. JELLIFFE.

163. UEBER CORTICALE BLINDHEIT (Concerning Cortical Blindness). R. Gaupp (Monatsschrift für Psychiatrie und Neurologie, Vol. v. No. 1, p. 28).

A man of sixty-four years presented left homonymous hemianopsia. A few weeks later he had a very violent mental shock, and after a few days had bilateral hemianopsia without an apoplectic "insult." Cortical blindness was at first complete and light sensation was very imperfect. The pupils reacted to light. The ocular muscles were not paralyzed and aphasia was not noticed. Retrograde amnesia lasted for several weeks, and the mental disturbance was considerable. Hemiparesthesia and motor hemiparesis of central origin existed. Improvement of the symptoms occurred with the restoration of a very small central field of vision. No mind blindness was detected. Red was the only color perceived. The mental disturbance became much worse during an acute intestinal attack.

A number of cases of complete cortical blindness, with restoration of a small central field, are on record. Sometimes the blindness is permanent, but in no case has the central field been so small as in the case reported by Gaupp.

Gaupp thinks it is remarkable that in many cases of bilateral hemianopsia the disturbance of vision has occurred without an "insult," and often has required several days for its full development. Frequently also failure of memory has been noticed. Disturbance of the power of orientation has been seen in bilateral disease of the occipital lobes, and loss of topographical sense has been found to be an important symptom of cortical blindness. The explanations given for this are not satisfactory. Usually the impairment in the sense of orientation occurs with impairment of memory. The formation of topographical images is a very complex mental process. Gaupp thinks his case shows that impairment in the sense of orientation need not be proportional to the impairment of cortical vision, for the former was slight in comparison with the latter. SPILLER.

164. DIE HYSTERISCHE TAUBSTUMMHEIT (Hysterical Deafmutism). Veis (Münchener medicinische Wochenschrift, 1899, No. 13, p. 415).

Hysterical deafmutism is a rare condition, since only about twenty authentic cases are on record. To the list the author adds the follow-

ing: A man twenty-six years of age, an innkeeper, of good family history and having previously presented no nervous symptoms, on awaking one morning was found to be deaf and dumb. He could neither hear nor speak, but could converse by writing, and no anesthesia, paralysis or other symptom of hysteria could be discovered. The condition had persisted nine weeks when he came under the care of the author. A cure was promptly effected by conveying in writing to the patient the suggestion that he would soon hear again, bringing forward with considerable parade the instruments for Eustachian catheterization, and inflating through the catheter first one and then the other ear. As the right ear was inflated the patient sprang up and said in a whisper that he could hear in that ear, and as the same procedure was applied to the left, he shouted that he could hear everything, and no more treatment was necessary. In speaking of the differential diagnosis, the author calls attention to the fact that in hysterical deafmutism writing, as a rule, is not interfered with, while in speech disturbances, due to focal lesions, agraphia to a greater or less degree is apt to be present. ALLEN.

165. EIN BEMERKENSWERTHER FALL VON SOGENANNTER GALOPIRENDER PARALYSE (A Noteworthy Case of Galloping Paralysis). Brassert (Allgem. Zeitschrift f. Psychiatrie, 55, 1899, 5).

This case occurred in a man of forty years of age. He had always been a healthy man. He was taken ill, and in a few weeks developed a typical case of general paresis, with well marked expansive ideas and delusions of grandeur and of power. He was removed to the asylum, and died there after ten days from a series of convulsive seizures, which numbered 99 in twenty-four hours.

The autopsy showed a marked hyperemia of the meninges, atrophy and soggy edema of the left hemisphere, and throughout the brain marked degenerative lesions indicative of grave chronic alcoholic intoxication. JELLIFFE.

166. ZUR GENAUEREN LOCALISATION DER KLEINHIRNTUMOREN UND IHRER DIFFERENTIALDIAGNOSE GEGENÜBER ACQUIRIRTEM CHRONISCHEM HYDROCEPHALUS INTERNUS (Contribution to More Exact Localization of Cerebellar Tumors and their Differential Diagnosis from Acquired Chronic Internal Hydrocephalus). Rudolf Schmidt (Wiener klin. Wochenschrift, No. 51, 1898, p. 1170).

Schmidt reports two cases of cerebellar tumor. One patient had nausea and vomiting when she laid upon her right side. An angiosarcoma was found in the left cerebellar lobe. The other patient had vomiting, vertigo and ringing in the ears when lying on the left side. A glioma of the right cerebellar lobe was found. The tumors in these cases were believed to have compressed the vena magna Galeni or the aqueduct of Sylvius, and to have caused increased intracranial pressure. It is probable that vomiting, vertigo, etc., depending on a lateral position of the body, are symptoms of large tumors near the median line. These symptoms point to disease of the posterior cerebellar fossa. They indicate asymmetry of the intracranial process, and, therefore, are against the diagnosis of idiopathic hydrocephalus (meningitis serosa chronica), or of cysticercus situated mediately in the aqueduct or fourth ventricle, or of hysteria. They are especially valuable in connection with other symptoms of cerebellar growth. Lost knee-jerks speak more for tumor than for idiopathic hydrocephalus. SPILLER.

167. MYSTICISME ET FOLIE (Mysticism and Insanity). A. Marie (Archives de Neurologie, 8, 1899, p. 33).

The author gives a historical introduction showing how, in the eighteenth century, epidemics of religious delirium replaced those of

a demoniac form. At those times, cases of illuminism were frequent, Joan of Arc being an instance. In the middle ages, Christianity triumphed over old polytheistic superstitions and new religions of a monotheistic conception were instituted. No more epidemics of lycanthropy were to be seen. God or his representatives haunted the minds of the insane. At those times, an army of prophets arose, and the writings of vulgar demoniacs were hard to differentiate from those of such men as Luther, Calvin or Jean Huss. Religion found its inspiration in fear. Before adoring his gods, man learned to fear them. Primitive religious ceremonies aimed only at calming the anger of some divinity. Victims were needed, and in order to save his own life, man sacrificed animals or his own fellow men. The gods were represented by bodies of waters, wild animals, clouds, etc., the naturalist's religion. Finally the symbol freed itself from the idol. They ceased to adore the animal, but began to venerate and adore its qualities, shrewdness and courage. Zoomorphism was only emblematical, being the mode of a higher conception, that is, a religious language. The gods became human (in a figurative as well as in a literal sense). The idols still had heads of animals, but the body was that of man, or inversely. True anthropomorphism gradually came into play. Primitive fetichism took the place of polytheism, then different countries adopted in preference such and such a divinity as more favorable to certain localities. Hence, the tendency to monotheism, to the belief of one powerful God, man's friend.

The insane, says Lemerie, ignorant of this principle of primary philosophy, imagined or created simpler hypotheses than those they refused to accept, taking up long abandoned theories and ideas. So long as it was believed that gods came upon the earth, Jupiter, Mercury, Apollo, Diana, Venus, were frequent visions to the insane. They thought they were married to Satyrs, to the god Pan, snakes and bulls. In the unreasonable conception of man's mind, angels and devils took the place of gods. Richet says that after a time, the devil lost some of his prestige, and the good angels took his place.

The author brings out in an interesting manner in what different way the two great divisions of Christianity—Catholics or Protestants—were disposed to this form of insanity. According to Marie, one's religion will greatly influence one's insanity. As a general rule, the Catholic is uneasy about his soul, is apprehensive of heavenly punishments, is afraid, in despair. The Protestant's mysticism is a pretension of understanding and explaining the symbols of Scriptures. Pride and prophetic exaltation are its leading features. One is a sinner, the other, a messenger from heaven. The Catholic is doomed, the Protestant, a prophet. According to our author, religious megalomania is found amongst Protestants, and religious melancholia amongst Catholics. It would be interesting to compile statistics upon this question in the asylums of France as well as in other countries. Superstition is present as an unconscious element in the normal brain, but unnoticeable, hidden, so to speak, by complete development of intelligence. Should the mind become affected in some way, then superstition may be a prominent feature. These deliriums have been defined by Meynert: "The appearance of a subconscious superstition in the developed brain."

The medico-legal points brought out are of interest. The insanities are two kinds: The first, divergent, *i. e.*, directed to hurt others; secondly, convergent, *i. e.*, upon themselves. Marié reports a few cases to illustrate his statements, and he comes to the conclusion that religious insanity is the most dangerous form to the patient and to society. On account of their suicidal and homicidal tendencies, they

should not be paroled or discharged from asylums. Religious delirium or insanity being very contagious, seclusion should be carried out in order to prevent spreading. These are the only efficient therapeutic measures for these cases.

JELLIFFE.

168. DIPHTHERITIC PARALYSIS. Francis Huber. (Pediatrics, 1899. Vol. vii, No. 11, p. 501).

In the majority of cases this condition must be attributed to the toxic effects of the Loeffler bacilli. It occurs in from 10 to 25 per cent. of cases, varying in different epidemics. The treatment of the original disease by antitoxin or otherwise does not appear to have any influence upon the subsequent development, nor does the type, mild or severe, determine its occurrence in any way. The difficulty in deglutition and the return of fluids through the nose occurring during the first week, due to mechanical interference with the functions of the velum by membrane or edema, must not be confounded with real diphtheritic paralysis. The latter, though it may occur as early as the seventh or eighth day, generally manifests itself during convalescence in the second or third week. In some patients the progressive muscular weakness of the body attracts attention, though no paralysis exists. Upon examination paralysis of the soft palate may be found with loss of patella reflex. The absence of patella reflex may be the only evidence of an implication of the nervous system; generally it is associated with paralytic manifestations. The paralysis is the result of a toxic peripheral neuritis—a parenchymatous degeneration of the nerves and not of central origin. Ordinarily the soft palate is first attacked—the nasal twang to the voice, perhaps regurgitation of fluid through the nose and some difficulty in deglutition are observed. If the pharyngeal constrictors are involved food enters the larynx and a spasmodic, croupy cough results. Many of these patients die of an aspiration pneumonia. Upon examination the soft palate appears relaxed and immovable, with diminished sensibility and absence of reflex. The extrinsic and intrinsic muscles of the eyeball are next involved, causing squint, ptosis and loss of accommodation. The paralysis may be confined to one limb; commonly both arms and legs are affected, or paraplegia may result. Frequently there is a weakness of the lower extremities and sometimes general muscular incompetency, but no real paralysis, with absence of tendon reflex. The danger is extreme when the respiratory muscles or heart are implicated. Heart failure may be rapid or come on slowly. The usual form appears during convalescence, at a time when all danger is apparently over. The accident may be the result of an infectious myocarditis or neuritis of the cardiac nerves, and sometimes of thrombosis or embolism. The bladder and rectum usually escape. In rare cases ataxia has been observed. Sensory paralysis may occur, in the form of anesthesia, amaurosis, or deafness. The prognosis is not as grave as one would infer from the condition in which many of the patients are found. A large number run a favorable course in from four to ten weeks. In the treatment of these cases absolute rest must be insisted upon. The general condition must be improved by food, air, tonics, and particularly iron in a digestible form. The remedy *par excellence* is strychnia in full doses, per month, or, in severe cases, hypodermically. Careful massage of the extremities will improve the cutaneous circulation and the condition of the muscles. If respiration is endangered, electricity must be employed in addition to artificial respiration, rythmical traction of the tongue and the subcutaneous administration of strychnia.

FREEMAN.

169. THE ETIOLOGY AND DIAGNOSIS OF CEREBRO-SPINAL FEVER. William Osler. (British Medical Journal, No. 2008, page 1517, June 24th, 1899).

The author first presents an excellent short review of the bacteriology of cerebro-spinal meningitis with the interesting results of his personal experience in a Baltimore epidemic. In fourteen of the sixteen cases in which lumbar puncture was made, positive results were obtained. In thirteen the diplococcus intracellularis was found, in the remaining case the presence of this organism was doubtful and a staphylococcus grew in culture. All of the six cases which came to autopsy showed mixed infection; that is, an examination of all the organs disclosed two to four kinds of micro-organisms, although the meninges may have contained only one. The author concludes that it is reasonable to suppose that the diplococcus intracellularis is the cause of the disease. "That a primary cerebro-spinal meningitis may be due to the pneumococcus is universally acknowledged; but it is in the highest degree unlikely that a remarkable specific affection like cerebro-spinal fever should be caused by two different organisms."

In treating of the diagnosis the author notes particularly the variability of all symptoms, the almost uniform presence of Kernig's sign and the great diagnostic value of lumbar puncture. What is said of Kernig's sign may be quoted in full.

"Described by a Russian physician, and studied in Germany and France, this interesting sign has not attracted the special attention of English and American physicians, though J. B. Herrick, of Chicago, at the last meeting of the Association of American Physicians, spoke of its value. It has been present in all our cases in which it has been looked for. It is, I think, an old observation that the subjects of protracted meningitis, particularly children, very often lie with the thighs flexed upon the abdomen, and with the legs in a state of partial contracture, so that they are with difficulty extended. To test for Kernig's sign the patient should be propped up in bed in the sitting position, then, on attempting to extend the leg on the thigh there is contraction of the flexors which prevents the full straightening of the leg. On the other hand, in the recumbent posture the leg can be fully extended. Many patients with meningitis are not in a condition to sit up, and the test can be equally well made by flexing the thigh on the abdomen, when on attempting to extend the leg, if meningitis be present, the limb cannot be fully extended. Friis found the sign in 53 of 60 cases, and Netter in 45 of 50. It is stated to be present in all forms of meningitis when the spinal meninges are involved. The presence of the sign is no indication of the intensity of the spinal involvement, as it existed in a very marked degree in a recent case of pneumococcic meningitis, in which there was no positive exudate on the spinal meninges, only a turbid fluid. Netter's explanation of the phenomenon is as follows: "In consequence of the inflammation of the meninges the roots of the nerves become irritable, and the flexion of the thighs upon the pelvis when the patient is in the sitting posture elongates, and consequently stretches the lumbar and sacral roots, and thus increases their irritability. The attempt to extend the knee is insufficient to provoke a reflex contraction of the flexors while the patient lies on his back with the thighs extended upon the pelvis, but it does so when he assumes a sitting posture."

Following the consideration of epidemic cerebro-spinal meningitis, the author describes in his usual lucid style the sporadic form of the disease and pneumococcic meningitis. We are unacquainted with anything on the latter subject at once so terse and so valuable.

PATRICK.

170. UBER DIE SYMPATHISCHE PUPILLARREACTION UND ÜBER DIE PARADOXE LICHTREACTION DER PUPILLEN BEI DER PROGRESSIVEN PARALYSE. (On the Sympathetic Pupillary Reaction and the Paradoxical Pupillary Light-Reaction in Progressive Paralysis). Joseph A. Hirsche. (Wiener klin Wochenschrift, No. 22, June, 1899, p. 592).

This paper is based on a study of the entire material afforded by the psychiatric clinic of Krafft-Ebing for a period of three years. The clinical research was carried on with almost painful attention to technique, and in several instances supplemented by necropsic examination of the cervical sympathetic and its ganglia, together with the upper portion of the spinal cord.

The author defines the sympathetic pupillary reaction as a dilatation of the pupil on irritation of sensory nerves, and the paradoxical pupillary reaction as a dilatation of the pupil on the approach of a light. In the case of the first named the irritation was brought about by pricking the cheek with a needle, by pinching it or by electrical stimulation. The conclusions arrived at are as follows: 1. In the presence of an Argyll-Robertson pupil the sympathetic pupillary reaction is practically always absent, and its absence almost without exception antedates the failure of the light reflex. 2. This failure of the sympathetic pupillary reflex has as its pathologico-anatomical basis, atrophic alterations in the cervical sympathetic and its ganglia, and also isolated atrophy of the lateral cell-groups in the lower cervical and upper dorsal cord. 3. The so-called paradoxical pupillary light reflex occurs: *a* with the Argyll-Robertson phenomenon, if the sympathetic reaction is fully retained; and *b* with the Argyll-Robertson phenomenon and absent sympathetic reaction, if in connection with an insufficiency of the internal recti outward rotation of the bulb occurs on illuminating the eye. In Hirschl's opinion the term paradoxical cannot properly be applied to either of these latter phenomena, because he believes that in the first the pupil is dilated by the warmth of the illuminating body through the retained sympathetic reaction, and in the second, by the outward rotation of the bulb. The value of the absence of the sympathetic pupillary reflex is regarded as minimal from a differential standpoint both for the reason implied in conclusion number one, and because its absence has been noted in chronic alcoholics and in persons after the age of sixty.

COURTNEY.

171. GASTRIC CRISES AS AN EARLY SYMPTOM IN TABES. Pineles. (Wien med. Club, in Wiener klin. Wochenschr., Dec. 29, 1898, p. 1212).

For a period of two years, a man, aged 32, and having a syphilitic history had attacks of gastric cramps and bilious vomiting which persisted one or two weeks. These attacks recurred at shorter and shorter intervals, but on the patient's entry into the hospital in September, 1898, no tabetic symptoms had manifested themselves, though later on he suffered from lightning pains, Romberg's sign, inequality of the pupils, tardy reaction, etc. After each crisis the cutaneous anesthetics and paresthetics were noted.

The second case was that of a woman, of 41 years, who for two and a half years had been subject to violent gastric pains and vomiting, which recurred at longer or shorter intervals. An explanatory laparotomy was even done, but the symptoms continued unrelieved. In October, 1898, aortic insufficiency was discovered and loss of the pupillary reflex took place.

Thus in both cases the gastric symptoms put in an appearance from two to two and a half years before the more usual signs could be noted.

JELLIFFE.

Book Reviews.

LA PUBERTA STUDIATA NELL'UOMO E NELLA DONNA IN RAPPORTO ALL' ANTHROPOLOGIA, ALLA PSICHIATRIA, ALLA PEDAGOGIA ED ALLA SOCIOLOGIA. By Dr. Antonio Marro, Medical Director of the Royal Asylum of Turin and Docent in the Regia Università. Fratelli Bocca, Turin, 1898.

The medical world is constantly brought face to face with the fact that for a number of years past a renaissance in medicine has taken place in Italy. In the fields of neurology, psychiatry, and anthropology, the work produced by these investigators has partially revolutionized these sciences, and in the special line of work of which this present volume is a representative it might almost be claimed that the Italian students have made the most important contributions. Certainly, this present work of some 500 pages will remain a classic for years. The work is divided into twenty chapters, in which the natural history, both normal and abnormal, of the great physiological period of human nature, puberty, is carefully studied. The opening chapters give a series of valuable comparative studies of the physical development in man and woman, including the changes in the bodily form, the voice, the changes in special senses, and in the psychical and sexual experiences. These chapters abound not so much in citations from literature, though such bibliographical research is not neglected, but in extensive observations, for which the author has had exceptional opportunity, as he outlines in his preface. Chapter four divides the period of puberty in three epochs, and the author discusses the various experiential crises that arise in these. Chapters five, six, seven and eight take up some of the abnormalities noted during this period, the physical and mental character of the degenerate, the alterations in the generative instinct, and the various psychoses of puberty being exhaustively treated. These latter he discusses under three or four heads. He shows how the psychoses (1) of puberty, as a predisposing cause, (2) due to the evolution of the generative instinct (3) of the period itself, and, finally (4), of this period, but having no relation to the process of development, are all intimately related one to the other, and that hard and fast lines of distinction in the different types are not to be laid down. The catatonia of Kahlbaum is dwelt upon as a type of the later period of genital development. The following four chapters, nine, ten, eleven, and twelve, treat of a number of interesting topics that can hardly be summarized. They include: the effects of abnormalities of the sexual organs on the physical and psychical life; eunuchs, male and female; glandular and bodily metabolism and the influences of ablation; physiological changes accompanying degeneration of the organs, and the causes of sexual degeneration and its relation to other forms of degeneracy; and, finally, the influences of alcohol, of physical surroundings, of hunger, cold, and the emotions, on degeneracy. These subjects are all carefully handled, and the conclusions reached seem sound. The remaining eight chapters in the book must claim careful attention, for here are to be found a large number of observations on the moral hygiene of youth in all of its various aspects. Physical hygiene, feeding, early sexual hygiene, gymnastic exercises, various sports, and even dancing, are treated of in the thir-

teenth chapter. The hygiene of study, mental work in general, the relations of the period of puberty to mental work, what books should be read, what kind of ideas should be inculcated, what special philosophies taught, are discussed in the next chapter on hygiene of the mind. The hygiene of the morals is also taken up by our author in a way that is eminently practical. He, for instance, discusses the advantages of the methods of reward and punishment for certain classes of social delinquencies, and shows that there are differences in men and women with reference to the actions of these two means of accomplishing discipline. They also vary during the period of puberty. The marked hyperesthesia accompanying the period should be borne in mind by parents, whereby combativeness is developed out of all proportion to the cause, when punishment is used as a means of correction. This chapter, as well as the next, would prove of excellent service to over-zealous and under-instructed moral reformers, as well as to parents and teachers, who have much to learn about youth at this critical point in their life-history. In the remaining chapters many similar problems of pedagogy, general and municipal sociology and race psychology are treated with careful discrimination and with practical import. How can persons and houses of reformation be made most effectual in our social melange? What are the best ways of handling the various neuroses and psychoses of puberty? How, in accordance with modern ideas, is the new factor of woman competition in the struggle for existence to be thought out? How shall the prostitute be regarded, and what can be done for the general subject of prostitution? These and many other most important problems are handled in a manner that is eminently just and conservative. The author does not incline towards didacticism; he has no pet theories of general reform, but he has the true philosophical spirit and has given an accurate and temperate series of studies and conclusions. We hope to see the volume translated, that it may reach a wider circle of workers in medical sociological lines.

JELLIFFE.

SUGGESTION UND IHRE SOCIALE BEDEUTUNG. W. von Bechterew.
German translation by R. Weinberg. Verlag von Arthur Georgi,
Leipzig, 1899, p. 85.

In this latest contribution to the study of suggestion, the subject is treated not so much from a purely medical as from a sociological and psychological standpoint. The influence of suggestion in determining the actions of the individual or the masses for good or evil finds a place under a somewhat different terminology in many works on psychology. Considered, however, in the light of suggestion, many themes which seem hackneyed take on a new interest. The dancing epidemics of the Middle Ages, the crusades, the salons of Mesmer, the Paris Communes, receive a careful analysis in this work, and the various religious crazes and faith cures of the present age also are studied. The greater part of the book is taken up with a critical study of a religious craze which came under the author's observation in Russia, and whose leader, a paranoiac—Muljowanny—was under his care in the insane asylum at St. Petersburg. The very thorough examination of this case and the analysis of the epidemic will interest not alone the neurologist, but the psychiatrist and psychologist as well.

We should feel indebted to von Bechterew for a definition of suggestion, which frees it from its narrow connection with hypnotism. Suggestion, he states, is a special mode of influencing an individual, with or without his knowledge, by a second person acting intentionally or unconsciously.

The translation from Russian into German will be a disappointment to those who are accustomed to the easy and natural style of such writers as Strümpell. McCARTHY.

ARBEITEN AUS DEM INSTITUT FÜR ANATOMIE UND PHYSIOLOGIE DES
CENTRALNERVENSYSTEMS AN DER WIENER UNIVERSITÄT. HERAUS-
GEGEBEN VON PROF. DR. HEINRICH OBERSTEINER. HEFT VI, FRANZ
DEUTICKE, LEIPZIG UND WIEN, 1899.

Some years ago Prof. Obersteiner began the practice of issuing at irregular periods and in the form of separate volumes the work done in his laboratory in Vienna. Six volumes have now appeared, and each is a most creditable production. We miss in this latest the names of some of the contributors to earlier "Arbeiten," but the sixth volume is not inferior to those that have preceded it. Students of all nationalities owe a debt of gratitude to Prof. Obersteiner for the privilege of working in his laboratory, so generously offered, and his students scattered throughout civilized lands, as well as other persons, view with pleasure the progress manifested by the "Arbeiten."

The sixth volume is formed by six original papers, and is well illustrated. The first paper, by Spitzer, contains the description of a solitary tubercle in the floor of the fourth ventricle, and of the degeneration caused by this growth; and a long discussion on paralysis of associated ocular muscles (*Blicklähmung*). Special attention is paid to the posterior longitudinal bundle. In Spitzer's case, both fifth nerves were found degenerated, although they were not directly involved in the tumor; they were supposed to have been pressed by the tumor against the base of the cranium, or to have been injured by the distortion of the pons caused by the growth of the tumor. The possibility of symptoms produced by injury of structures at a distance from the seat of a new growth must always be borne in mind in localizing a tumor within the central nervous system. Degeneration observed by Spitzer in the upper part of Burdach's columns is explained in an ingenious way. Pressure by the tumor was believed to have been exerted on the first cervical roots, and to this pressure and the resistance offered by the ligamentum dentatum the degeneration of these roots was ascribed. Degenerated fibers found in the anterior columns of the cord and terminating in the anterior horns of the cervical region were believed to have had their origin in the interbrain or midbrain, in Deiters' nucleus, and in the nuclei of the posterior columns, possibly also in the cerebellum. Spitzer describes two tracts under the names of ventral and lateral tegmental bundles.

Zappert gives a lengthy description of the degeneration in the spinal cord of children, to which he has called attention in a previous paper. Degeneration of the intraspinal portion of the anterior roots, of the accessorius, and of the fibers issuing from Clarke's columns and passing to the direct cerebellar tracts, and of the motor bulbar roots, are not rare findings in children under two years of age. Alteration of the posterior roots and of the spinal white matter is more uncommon and less intense. The pronounced changes in the spinal cord, as seen by the Marchi method, were believed by Zappert to be pathological, and were found only after severe diseases of long duration. It seems that the anterior spinal roots of young children are especially vulnerable. In rare cases the motor cells in the anterior horns were also found altered. The changes in the anterior roots may have some relation to the convulsions of childhood, but the full significance of this degeneration is unknown.

We have from Neurath the report of the case of a child who had

been normal until hemiplegia developed after scarlet fever. Numerous sclerotic areas were found in the brain. The glia was altered in these areas, and large cells with large nuclei and processes, resembling ganglion cells, were found within and without these sclerotic foci. These areas were not the result of the scarlet fever, as the time intervening between the attack of scarlet fever and death was far too short for their formation, and no vascular lesions were observed. The hemiplegia was supposed to have resulted from the action of the poisonous products of the scarlet fever on the nerve cells.

Kure's investigations on the trigeminus nerve of the rabbit show that the cells of the cerebral root of this nerve do not conform to the type of the motor cells of the cord, but resemble those of the Gasserian and spinal ganglia; they have, therefore, probably a different function from the cells of the motor nucleus of this nerve. The fibers of the cerebral root, and those arising in certain cells of the locus cœruleus and entering the portio minor, and fibers of the motor root, do not decussate.

Pineles reports a case of tubercle of the left cerebellar hemisphere, and one of glioma of the left cerebellar hemisphere. He enters into a long discussion concerning the functions of the cerebellum and refers to the work of a number of investigators. His paper is largely in the form of a critical digest.

A very interesting case of congenital defect of the brain is described by Zappert and Hitschmann. The child lived eleven days. The spinal cord was well formed, except that the pyramidal tracts were absent and some degeneration existed in the posterior columns. The anterior corpora quadrigemina, the cerebral peduncles, the basal ganglia and parts above these were entirely absent. The eyes were developed, but the optic nerves were atrophic. It is interesting to note that the cells of the anterior horns of the cord appeared to be normal by the carmine stain, and that the absence of the cerebral impulses transmitted by means of the pyramidal tracts was not sufficient to arrest the development of these cells. A peculiar degeneration of the posterior columns in the uppermost part of the cord could not be satisfactorily explained. It is certainly a noteworthy fact that a human being may live a certain time without a cerebrum—in this case, eleven days.

SPILLER.

A new journal devoted to psychiatry is just announced. It is entitled "*Psychiatrische Wochenschrift*," and is to be devoted to the reporting of mental diseases and general correspondence. Its editors are Alt, Anton, Guttstadt and Mendel; acting editor, v. Bresler; publisher, von Marhold, in Halle.

The new journal is to be a quarterly and its price 4 marks.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

ISOLATED FINGER PARALYSIS.*

By WILLIAM BROWNING, M.D.,
BROOKLYN, N. Y.

The title given is so far inexact, as in the present case only the extensors were affected and not the flexors.

The gentleman, O. P. A., whose case I have to relate, was 78 years of age at the time of his death in December, 1897. He had been a prominent man in one of the New England cities, and had passed an energetic though rather easy life. His usual weight was about 200 pounds. In general, he was a good liver, though an abstainer from all alcoholic drinks. Since youth he had been a chewer of tobacco, never a smoker. For many years, and it was claimed even since childhood there had been an increasing irregular tremulousness of the hands, appearing only on effort; apparently, however, this was of the senile form. Otherwise, his health had always been excellent.

Perhaps it may be as well to begin with the notes made when the photographs were taken in November, 1894. His mental condition was then clear and good, though he no longer felt equal to business affairs. The skin of the fingers was heavily wrinkled, as is apparent from the pictures. During an attack of bronchitis a year previously, there had been moderate albuminuria; but this had since disappeared. His urine at times showed an excess of uric acid, though he had never had frank gout. Beyond this and the marks of age there was no further trouble, constitutional or otherwise. He was right-handed.

His special trouble began some three years before this.

* Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

i. e., when he was 72 years of age. He had always been a follower of the rod and gun. One day while trout-fishing there was a sudden snap of something at the back of the right hand as he was swinging the rod. He immediately found that there was complete extensor paralysis of the middle finger, passive motion being still free. This was promptly followed by severe pain in the back of the hand, extending partly down that finger and up the forearm. The pain abated considerably within a couple of hours, and by next day was wholly gone. After that he learned to extend this finger by getting the two on either side under it. This soon became so habitual as scarcely



Fig. 1]

to be noticed by others. It may be added that there had been no previous trouble with, or accident to, the part; except for the strain on the fingers of handling a fishing-rod, the occurrence was spontaneous.

The above described condition continued unchanged for a year and a half. Then, in addition, the adjoining ring finger of the same right hand became affected in like manner. This occurred while lifting a small box. Hence each attack occurred at times when the muscles were presumably under considerable strain. This second involvement was also immediately

followed by local pain, even more intense than the first. It started from the back of the hand and extended up the forearm to the elbow. Bathing and a somewhat firm bandage to hand and forearm eased the suffering by evening. Yet the pain and some soreness locally lasted into the following day at least. Since then there has been no change.

No pain nor premonition preceded either attack. Sometimes the fingers seem to get caught, and he has to start them with the help of the other hand. Passive motion is free and natural in all directions. Grip (*i. e.*, flexion) does not appear to be interfered with. There was no chance to test the electrical reactions. The local changes at this time were so slight in appearance as scarcely to be detected. In comparison with the other side there was just a faint depression over the tendon-



Fig. II.

path at the back of the hand; and, when flexed, a slight projection of tendon over the ring-knuckle (aspect towards finger). The position of the hand as a whole is well shown by the accompanying pictures. It might be added that there was no anesthesia, subjective or objective. No difference in the wrist-reflexes—all for that matter slight.

As to the presumptive seat of the lesion in this case, the fact of severe local pain at each attack was sufficient to exclude central trouble. This was also borne out by the exceedingly limited and complete character of each paralysis. Neither

were the symptoms those of even a local neuritis, though some gouty affection could not be entirely excluded. My own conclusion at the time was that there must have been a fraying-off of the tendon.

While his subsequent history has no direct bearing on this matter, it may be briefly summarized. The hand paralysis remained entirely unchanged.

In 1895 the right eye showed signs of beginning cataract, though it never progressed much. Slight puffiness appeared about the feet and legs. He twisted the right foot (possibly a slight stroke), and thereafter it was more swollen and the toes, especially the big toe, showed a flexion-contracture. The right knee-jerk was a little the stronger (no clonus), and the right leg was more sensitive to pressure. There was also a very slight contracture at the right knee. From this time he gradually became feebler, his mental and physical strength failed, and he died of senile exhaustion some six years after the first onset of his finger paralysis.

Autopsy, December 31, 1897. Body somewhat emaciated. Only the upper extremities examined.

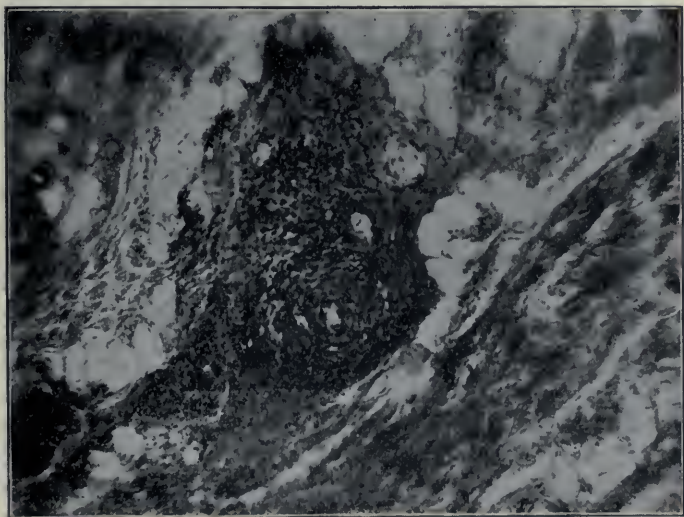
Maximum circumference of left forearm 23.5 cm., of right 23.0 cm. On laying bare the deeper structures of the forearms, marked changes were evident on the right side, the left proving normal.

On the right the long extensors of the middle and ring fingers showed analogous changes. Their tendons over the distal three-fifths of the dorsum of the hand were approximately natural. But from there to about one-third up the forearm—a hiatus of perhaps four inches—these tendons had so far disappeared and become amalgamated with their thickened sheath that only a strand could be separated out as the possible remnant. In fact this strand, as contrasted with the glistening white tendon, had the appearance of pale sheath-tissue. The agglutination and thickening were most marked in the region corresponding to the annular ligament of the wrist. On subsequently cutting through this material, a small lump of muscular tissue was found in the middle of it. The tissue proper of the two affected muscles was much shrunken, disintegrated, separated into fragmentary masses, and flecked with scant patches of fat. The belly of each muscle appeared to be replaced by gravish-yellow fat-tissue. Close to the elbow, however, some of the muscle-fibers were preserved.

The adjoining muscles and tendons—to index, little finger and thumb—were intact, except that the index tendon was enveloped partly in the sheath-thickening above described.

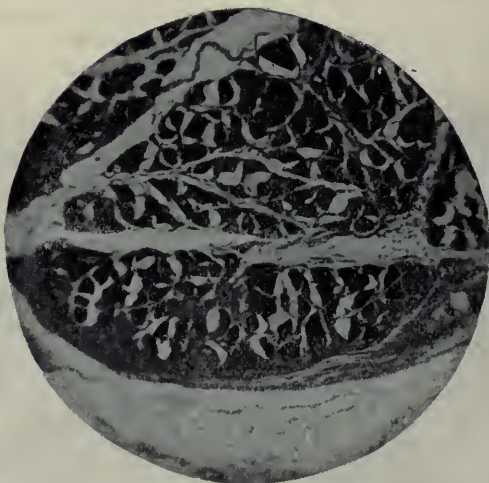
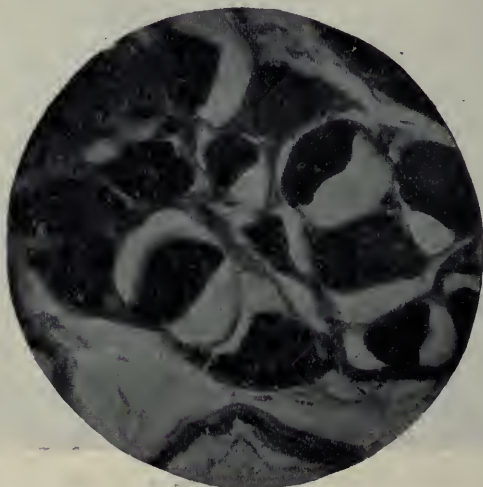


Specimen No. 1.



Specimen No. 4.

Report by Dr. Archibald Murray, Fellow of the Hoagland Laboratory, on the findings in the specimens of muscle submitted by Dr. Browning.

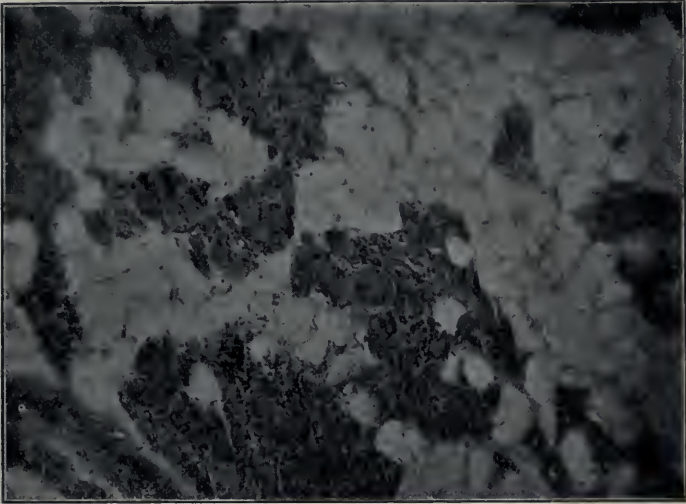


Specimen No 4 under different magnifications.

"Specimen No. 1. This is taken from a mass of degenerated muscle from one of the two involved muscles, near the tendon.

"Many of the fibers have undergone retraction, their course being very irregular. Hyaline degeneration is well marked in many of the fibers.

"Specimen No. 4. This is from a mass taken from the back of wrist. In the portions of this specimen where the



Specimen No. 8.



Specimen No. 10.

muscle fibers have been cut transversely, they appear to have atrophied or shrunk to about one-half their normal size, the

remains of the muscle fiber occupying one-half of the space surrounded by the endomysium, the other half being empty. Some of the atrophied fibers show a well-marked hyaline degeneration. There is a moderate small-round-cell infiltration, and the vessels show a well-marked obliterating endarteritis.

"Specimen No. 8. This is taken from the belly of the affected muscle, near the elbow joint. (Extensor communis digitorum.)

"The small-round-cell infiltration is most marked in certain portions of this specimen. Here and there are seen isolated masses of muscle-fibers surrounded by spaces from which the fibers have disappeared.

"Specimen No. 10. This is from one of the involved muscles and shows some irregularity in the size of the individual fibers."

Remarks:—Even with the aid of the post-mortem findings, it is not entirely clear what was the nature of the original process here. If we suppose a local inflammation, it hardly accounts for the practical disappearance of a portion of the tendons. There had been no suggestion of trigger-finger, nor would that in any way explain the facts. Though the neighboring tendons as well as those of the opposite side looked perfect and of unusual strength, still the character of the original attacks and the condition found on autopsy speak for a rupture of the tendons. At such occurrence, some effusion must have developed, and this explains the slight thickening at the wrist and the partial enclosure of the index tendon. The repetition of the attack shows that it was something more than an accidental occurrence. In any case it was a local affair, and doubtless in some way senile in character.

Examination of the special journals and text-books, and even the *Index Medicus*, has so far failed to disclose any case at all corresponding to the present one. Dr. Fletcher of the Surgeon General's Library also very kindly looked the matter up, but found no reference bearing directly on this case.

Paralysis of a nerve of the arm is well known; spasm of the muscle of one or more fingers is occasionally described; occupation neuroses in great variety are recognized; joint affections causing stiffness of one or more fingers are also now and then met with (*e. g.*, Prince's case in *Bost. Med. and S.*

Jour., 1899, March 2, involving middle, ring and little fingers, with weakening of the flexors); trophic alterations may occur.

The nearest analogy, however, of the present case is furnished by the rare cases of traumatic rupture of finger tendons.

Second¹ reports a case of subcutaneous rupture of the extensor tendon of phalanx of little finger.

Busch² gives one of the thumb, three of the little finger, and one of the ring finger.

Polaillon³ is said to mention two cases, one of the index and the other of the little finger.

Mueller⁴ reports a case.

M. Gangolphe⁵ reports the case of a man of 35 years who had subcutaneous tearing of extensor tendon of the second phalanx of the thumb, due to violent flexion. Pain, swelling, ecchymotic discoloration, position of phalanx in partial flexion, and increased lateral motion resulted.

Hence while in one or the other of these cases, each of the fingers has been implicated, still they refer chiefly to the extensor tendons of the little finger. So far as the subject has been looked up, however, these were all due to external violence, and have no further bearing on the case here reported.

DISCUSSION.

Dr. F. X. Dercum said that the problem brought forward by Dr. Browning was an unique one, as we know little about the senile changes that take place in the tendons. The thought suggested itself to Dr. Dercum that a break or tear of the tendon was produced by the muscular effort, and that the muscular changes were the indirect result of this injury.

Dr. Browning said that the pathologists were inclined to attribute the changes to the obliterating endarteritis; if this view was correct, he failed to see why similar changes were not found in the index finger of the same hand, inasmuch as obliterating endarteritis existed here.

¹ Bull. de la Société anat., 1879, p. 724.

² Centbl. f. Chirurgie, 1881, No. 1.

³ Art. "Doigt" in the Dictionnaire encyclopédique.

⁴ "Cas de rupture des tendons fléchisseurs du médius gauche." Mem. Soc. de Méd. de Strasb. (1881-2) 1883, XIX, pt. 2, 50-52.

Mem. et compt.-rend. Soc. d. Sc. Méd. de Lyon (1885), 1886, xxv pt. 2, 74-77.

ASTHENIC BULBAR PARALYSIS.*

BY WHARTON SINKLER, M.D.

This peculiar disease was first described by Wilks, in 1870, in *Guy's Hospital Reports*¹, where he relates two cases which he speaks of as "an unusual form of glosso-labio-laryngeal paralysis." In 1877² he described another case of disease of the medulla, in which death supervened within a few weeks from respiratory paralysis, and a careful autopsy revealed no perceptible lesion. In 1878, Erb³ published three cases, in which the following symptoms were peculiarly prominent:—bilateral ptosis, isolated in two of the cases, and in the third the intrinsic muscles of the eyeballs were also affected. There was weakness of the muscles of mastication, paresis of the neck muscles, and weakness of the tongue and limbs. The upper branches of the facial nerve were affected, while the lower remained intact. There was no muscular atrophy. The parietic symptoms fluctuated in intensity, improving by rest, and becoming aggravated by fatigue and exertion. Erb called the disease "A new syndrome, probably of bulbar origin." He was evidently not acquainted with the publication of Wilks' papers. Nothing more was added to the subject until 1887, when Oppenheim⁴ reported a case with autopsy. Soon after this Goldflam⁵ published a paper, in which he collected all previous cases, and added four new ones. To Goldflam belongs the credit of having first called attention to the importance of the fact that the morbid phenomena in this affection are essentially variable in intensity, and are rather due to peculiar weakness of the muscles, evidenced by their quickly giving out as a result of the slightest effort, than to general paralysis. In 1892 Hoppe⁶ wrote a paper, giving a full and complete account

* Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

¹ Wilks, *Guy's Hospital Reports*, 1870, Vol. XXIII.

² Wilks, *Guy's Hospital Reports*, 1877.

³ Erb, *Archiv für Psychiatrie*, Vol. IX, p. 336.

⁴ Oppenheim and Siemerling, *Berl. klin. Wochenschrift*, 1886, Vol. XXIII, p. 791.

⁵ Goldflam, *Deutsche Zeitschrift für Nervenheilkunde*, Vol. IV.

⁶ Hoppe, *Berliner klinische Wochenschrift*, 1892.

of the disease, and from this time more attention was drawn to it. Strümpell⁷ in 1895 reviewed the literature of the subject, and collected 21 cases which had been reported. Various names have been given to the disease, some of which are "Bulbar Paralysis, without Anatomical Lesions at Autopsy;" "Myasthenie grave pseudo-paralytique" (Jolly); "Syndrome d'Erb et Goldflam;" "Syndrome de Hoppe et Goldflam;" "Maladie d'Erb," "Syndrome bulbaire d'Erb;" "Chronic Progressive Bulbar Paralysis (Oppenheim).

Strümpell has called the affection "Asthenic Bulbar Paralysis," and this seems to have found general favor, as it is the name now generally used. In addition to the 21 cases collected by Strümpell, there have been 13 others reported. Of these, but three were by Americans—one by Collins⁸, one by Wheaton⁹, and one by Berkley¹⁰. The other cases recorded were by Marie¹¹, Ballet¹², Cardarelli¹³, Finizio¹⁴, Widal and Marinesco¹⁵, Silbermark¹⁶ (2 cases), Brissaud and Lantzenberg¹⁷ (2 cases), and Charcot and Marinesco¹⁸ (1 case).

The principal features of the affection are as follows: The disease appears usually before thirty, and may occur as early as the age of 12 or 15 years, with perhaps a slight preference for the female sex. It begins gradually and without apparent cause, but there may be quite rapid increase in the symptoms, and it may result fatally within a few weeks. Occasionally, there is at first pain, never very violent, and mild vertigo. An

⁷ Strümpell, *Deutsche Zeitschrift für Nervenheilkunde*, Vol. VIII, p. 16.

⁸ Collins, Joseph, *International Medical Magazine*, April, 1896, p. 203.

⁹ Wheaton, *Boston Medical and Surgical Journal*, 1898, p. 54.

¹⁰ Berkley, *Johns Hopkins Hospital Reports*, 1897, p. 94.

¹¹ Marie, *Bull. et mem. soc. méd. des hop. de Par.*, 1898, 3 S., XV, 437.

¹² Ballet, *Gaz. hebdomadaire de méd.*, Par., 1898.

¹³ Cardarelli, *Clin. méd.*, Pisa, 1898, IV, 81.

¹⁴ Finizio, *Riforma med.*, Napoli, 1898, XIV, Pt. 1, 589.

¹⁵ Widal and Marinesco, *Bull. et mem. soc. méd. des hop. de Par.*, 1897, 3 S., XIV, 518.

¹⁶ Silbermark, *Wiener klinische Rundschau*, 1896, X, 767.

¹⁷ Brissaud and Lantzenberg, *Arch. gen. de méd.*, Par., 1897, I, 257.

¹⁸ Charcot and Marinesco, *Compt. rend. de la soc. de biol.*, March 1, 1895.

early and most important symptom is the rapid fatigue usually shown in those muscles whose nerve centers are in the medulla, pons and crura. The first symptom may be drooping of the lids and diplopia, or else difficulty in mastication, speaking or swallowing. Involvement of the trunk and limbs usually occurs later, although the disease may begin in these parts. There are seldom muscular twitchings observed even in the tongue. When the disease is fully developed, there is permanent paresis in the upper lids and all the muscles innervated by the seventh nerve, and in the muscles of mastication. In the muscles elsewhere there is great and rapid fatigue after the slightest effort, which, in the later stages, amounts almost to complete paralysis. This is noted especially in swallowing, chewing and speaking, and it is also true of the movements of the limbs. The permanent paresis is found in those muscles which are continually in tonic contraction, and have little rest; that is, in the levatores palpebrarum, the muscles of expression, and the masseters. In some cases permanent paralysis of the muscles of the eyeballs is present, and these are also in constant tonicity; often, however, these muscles are entirely spared. Of the muscles below the head, only those of the back of the neck have been found permanently parietic, the explanation for this being, that they are in the same condition of tonic contraction as the muscles of the lower jaw. Ptosis is often the first symptom, and when the muscles of the eyeballs escape, it is important as showing an involvement of one branch only of the third nerve. Reaction of the pupils to light and accommodation always seems to be normal. The extremities are always involved, but weakness of the muscles of the organs of respiration, if it occurs at all, appears late, and the sphincters have never been found affected. On certain days the patient has more power than on others, but anything causing unusual fatigue or depression impairs the strength of the muscles. For example, in the case related by Wheaton, and in my own case, during the menstrual period the paresis was always greater. Irregularity of the heart's action, and tachycardia are sometimes met with. Sudden relapses are common, and may occur even one or two years after apparent recovery.

About fifteen autopsies have been made, and in none have

microscopical changes ever been found in any of the tissues, and from the clinical conditions gross lesions could not be expected. Most writers suggest the theory of infection by toxic materials, although no one can say if the poison is elaborated in the body or taken from without. It is exclusively an affection of the motor system.

The same exhaustion is shown to the faradic current, which proves that the peripheral neuron is involved perhaps alone, although the disease may be muscular. The knee-jerk is prompt, and muscular atrophy does not occur, even after a long period.

The prognosis naturally is doubtful, but the patients usually have periods of remission, and are frequently apparently quite well for several years at a time. There are a number of essential differences between this affection and true degenerative bulbar paralysis. There is no real atrophy of the muscles, either of the tongue, lips or extremities, and there is no complete paralysis of the muscles of the lips producing drooling or drivelling of saliva. The patient is usually able to expose the teeth, pucker the mouth, and to whistle. The deep reflexes are preserved, and there is no change in electrical irritability, except that it becomes exhausted after the continuous application of the current. There are no disturbances of sensibility and the special senses are unaffected. If, however, prolonged stimulation of the special senses is persisted in, they are liable to become quickly exhausted. For example, sight and hearing become impaired after long use. In true bulbar paralysis there is seldom involvement of the third nerve, or the lower facial, or minor branch of the fifth nerve.

Amyosthenia, or weakness of the lower extremities, is not observed in bulbar paralysis, and the attacks of disordered heart's action and dyspnea, which have been recorded as frequently occurring in asthenic bulbar paralysis, are seldom met with in the former disease. As already pointed out, the most characteristic feature of asthenic bulbar paralysis is the readiness with which the affected muscles become exhausted upon use. The patient, whose case I shall record, talks quite distinctly and readily at first, but after a few minutes' conversation her voice becomes weak, high-pitched and nasal. In masti-

cation of food the muscles soon become exhausted, and the patient has difficulty in swallowing the bolus. The movements of the arms and legs also cause feebleness in a short time. The knee-jerks, which are active on first examination, soon become exhausted. Jolly and Murri¹⁹ have pointed out the peculiar influence of electricity upon the muscles. At first, to the faradic current, these respond energetically, but after the passage of the current for a short time, the response becomes very feeble or nil. This Jolly called the "myasthenic reaction."

The opinion of Strümpell and others as to the pathology of the disease is, that it is infectious in character, and that the presence of some poison in the system exerts its deleterious influence upon the groups of ganglionic cells in the medulla and pons. Nothing so far has thrown any light upon the causation of asthenic bulbar paralysis.

It seems to me that a fact in connection with the case which I am about to relate throws some light upon the pathology of the disease. This fact is, that two brothers of my patient had nystagmus and nodding tremor of the head, and two of her own children have been affected in precisely the same way. This would suggest that there was a hereditary predisposition to lesions in the nervous system, and that there existed a developmental defect in the motor tracts of the medulla and pons. We cannot help feeling that, as we study cases of this kind, and observe the history of heredity, how strong is the influence in the direction of hereditary transmission. The number of familiar diseases of the nervous system is increasing each year, and we are beginning to learn that a certain class of diseases is liable to attack individuals in whom there is a poorly constructed nervous system.

The possibility of hysteria being mistaken for this disease is quite great, especially when ptosis and diplopia are the only symptoms, and disappear and reappear without apparent cause. One should always be guarded in making a positive diagnosis of hysteria before carefully considering the possibility of asthenic bulbar paralysis. In the early stages of the disease under consideration, it might readily be mistaken for nuclear ophthalmoplegia, but in asthenic bulbar paralysis the paralysis

¹⁹ Cited by Brissaud and Lantzenberg, *Arch. gen. de méd.*, Par., 1897, Vol. I.

is not so complete, and after rest the muscles involved always regain a certain amount of power. Moreover, the muscles of speech and deglutition sooner or later become affected in the latter disease.

The following case typically exemplifies the description given of asthenic bulbar paralysis by Erb, Strümpell and others :

The patient, Mrs. H. B——, was brought to my clinic at the Orthopedic Hospital and Infirmary for Nervous Diseases by D. C. B. Hough, of Hamburg, Penna. The patient was 37 years of age and married. She was born in America of Scotch parents. Her father died of apoplexy, and her mother is living and well at 65 years. Her maternal grandmother was blind for years before her death. She has two brothers and four sisters who are living, and who are in good health, with the exception of one brother who has nystagmus and a rotary tremor of the head. One brother who died at 30 years of age also had nystagmus and head shaking. She has been married for 16 years. Her eldest child is a boy of 15, who, like the maternal uncles above referred to, has nystagmus and a rotary tremor of the head. He is very small for his age, and undeveloped. He does not look more than 12 years of age, but he is active and very intelligent. His mother says that he does the work of a man on the farm where they live. Her youngest child is five months old, and this one also has nystagmus. The remaining four children are normal. Her husband is living and healthy. There is nothing in the history to give rise to any suspicion of syphilis.

Previous History.—There is nothing notable in the early history of the case. The patient had the ordinary diseases of childhood in mild form. Seven years ago she had a light attack of typhoid fever, and a short time after this, without any exciting cause, she gradually developed ptosis, first in the left and then in the right lid. This remained for at least six weeks. She went to the Wills' Eye Hospital in this city, and under treatment the ptosis gradually recovered. The history taken from the records at the Wills' Eye Hospital states that the lids commenced to droop in September, 1892. On January 30, 1893, when she was admitted to that institution, there was complete ptosis. After six weeks' treatment in which mercury was administered, there was scarcely any drooping of the lids. In October, 1895, there was a similar attack, and her doctor wrote for advice, as she could not come to the hospital. In October, 1897, she returned with diplopia gradually progressing. There was then paresis of both interni, and faintly so of the superior

recti muscles. The fields at this time were much contracted. In 1898, except that there was occasional drooping in the lid of the left eye, which is especially marked on near work, nothing seemed wrong. Still apparent paresis of muscles.

A child was born in December, 1898. It was not a difficult labor. She states that, a few days after the birth of her child, her eyesight began to fail more rapidly, and a month later she could not swallow well. Then her hands and arms began to lose power, and she also had feebleness in the legs. She has



FIG. I. Asthenic bulbar paralysis. Shows paralysis of left superior rectus in looking upward.

never been confined to bed. The weakness in her throat muscles, difficulty in swallowing and enunciating have been the most advanced symptoms. She has no pain, but some distress in the lumbar region. She has always had control of the bowels and bladder. She has not nursed her baby, and has menstruated regularly since its birth. At each catamenial period all her symptoms are more marked.

On examination, the peculiar expression of the woman's face is noticeable. The eyes are partly closed; the brows being elevated so as to raise the lids to give sufficient vision to go about. The face is without expression and mask-like. Absence of the upper teeth give rise to a peculiar sinking of the upper lip. There is no paralysis of the facial muscles. The tongue is protruded straight. The lips can be separated so as



FIG. II. Asthenic bulbar paralysis. Face at rest.

to show the gums, and the mouth can be puckered into the position of whistling. Her speech is nasal, indistinct and whining. After speaking for some time, almost complete aphonia exists. The effort of talking soon exhausts the power of articulation, but after resting for a time she is able to enunciate quite distinctly. Deglutition is difficult. After chewing and swallowing for a short time, the effort of swallowing becomes great,

and sometimes liquids are regurgitated through the nose. On inspection the throat muscles appear flaccid. Her hearing is good, and the special senses are unchanged. The arms are weak and soon tire after exertion. She walks in a shuffling and awkward manner, and the strength of the legs soon gives out and the gait becomes more unsteady. The knee-jerks are preserved and apparently do not become exhausted on repeated testing. The sensation is unimpaired, both as to tactile and pain sense. There is no thermo-anesthesia. Electrically, all of the muscles respond to the faradic current, and although not tested extremely, faradic irritability is not exhausted, as was observed by Jolly and others. There is no atrophy of the tongue, nor, in fact, of any of the muscles, and there is no fibrillary tremors in any part. There is no kyphosis of the spine. Dynamometer, right, 50; left, 50. The patient suffers no trouble with her digestion. The bowels are regular. She sleeps well and has no headaches.

Dr. A. G. Thomson made an examination of the eyes and made the following report: "Partial ptosis in both lids; more marked in the left. Pupillary reactions normal. Almost complete paralysis of the left external rectus, and partial paralysis of the left superior rectus. The accommodation is normal. The discs and fundi are normal. Form and color fields are unchanged."

There is apparently no mental disturbance, and the patient is of cheerful and contented disposition in spite of her infirmities. The fatigue which occurs in the various muscles, both the muscles of the eye and throat, and those of the legs and arms, is markedly characteristic of the affection, and exhibits the distinctiveness of the disease from organic bulbar paralysis.

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172. STOMATITIS NEUROTICA CHRONICA (Neurotic Ulcers of the Mouth). By W. Knowsley Sibley (British Medical Journal, No. 1998, page 900, April 15, 1899).

These ulcers have been described by a number of authors under various titles. Their principal claims to be neurotic seem to be that they occur in neuropaths; are aggravated by emotional and mental disturbances; are peculiarly chronic and recurrent; and are not easily explained on any other theory. The present custom seems to be to label the unexplained "neurotic." The author reports three cases, all in women beyond middle age.

PATRICK.

ASTHENIC BULBAR PALSY, WITH REPORT OF A CASE.*

BY JOHN PUNTON, M.D.,

KANSAS CITY, MO.

A few months ago the case I am about to report was referred to me by Dr. L——, of Columbus, Kansas, for diagnosis. In the course of my examination I discovered that the history and symptomatology were unlike that of anything in my previous experience, although my reading suggested to me what I think the case really is.

In order to confirm my suspicions I called to my aid my colleague Dr. Burnett, to whose co-operation I am mainly indebted for this detailed report. It is not my desire, however, in reporting the case to claim anything new or original, but simply to call attention to, and to put on record, one more case of this relatively rare form of nervous disease of which we have considerable yet to learn from a pathologic standpoint.

It is only within the last two decades that this affection has actually been impressed upon us as a distinct neurologic entity deserving a differentiation from that of Duchenne's disease, or true bulbar palsy, although Wilks¹ in 1870 presented a clinical résumé plus a post-mortem study that was followed a little later by an analytical study of three cases by Erb, whose combined efforts to gain special recognition of the disease fell flat. Only after Oppenheim, a decade later presented his non-confirmatory microscopical findings, showing the symptom-complex to be without a distinct anatomic change, was the stimulation sufficient to produce the series of papers that followed, illy describing the condition and leading to confusion in the production of a wide-spread nomenclature. Really, not until 1892² did the disease receive special recognition, this being prompted by Hoppe's³ paper. Dr. Collins in 1896 speaks of a dozen cases on record in which post-mortem examinations have been carefully made. While our patient is still

*Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

¹ Wilks, Guy's Hospital Reports, Vol. XXII.

² Collins, International Medical Magazine, April, 1896.

³ Hoppe, Berliner klin. Wochenschrift, 1892, p. 332.

living, and therefore the case offers no pathological evidence, it is clear that the clinical analysis is sufficient to demonstrate wherein the case differs from a chronically progressive degenerative bulbar paralysis, characterized by fibrillary twitchings, atrophy, and paralysis; and that it is almost a typical picture of those cases of asthenic bulbar palsy as verified by necropsies. The case in point is as follows:

Family History.—Grandparents, dead; no knowledge of the cause of death. The mother of the patient is asthmatic; the father is a strong healthy man.

Personal History.—The patient, Mrs. B., is twenty-five years old, married happily, and the mother of one child three years old. Prior to marriage she suffered, at intervals, from attacks of so-called rheumatism, characterized by pain in the joints without swelling; otherwise she was an average healthy girl. Her life has been devoid of any special hardships or mental depression.

The symptoms of her present ill health date back about three years. Shortly prior to this time she gave birth to her first and only child. The parturient act was prolonged and difficult. A midwife officiated, leaving the usual lacerations as souvenirs of her skill. Operation twelve months later for lacerated cervix was done successfully, while she was being treated at Hot Springs for her supposed rheumatism. She believes that she has not been well since her delivery. The first manifestation of the condition now complained of was an affection of the eyes, coming on at intervals, when she would see double, and her vision would become more or less dim—a manifest weakness of her ocular muscles. Ptosis of left eye supervened, being also worse at intervals. Her general health began to fail, and amyosthenia was marked. She became profoundly weak on the slightest exertion, the extremities seemed like heavy burdens; mastication, which at first appeared normal, gradually became fatiguing, swallowing became difficult and imperfect, and articulation also became impaired. The voice changed in pitch, tone and quality, being distinctly nasal in character. The tongue became less dexterous and was the seat of "numbness," as she explains it. The prehensile acts performed by it during mastication were limited, imperfect and fatiguing. The food could neither be removed readily from the buccal grasp, nor be conveyed posteriorly to the constrictors, which in turn were functionally derelict, and deglutition, especially of solids, was markedly unsatisfactory. While abnormal weariness, prostration and rapid heart action are induced by slight efforts of bodily occupation, she has no morbid

fears, apprehensions, dreads or perverted conceptions; neither is she moody, despondent or given to morbid mentality. In two years from the onset of the disease she had ptosis of the right eye and weakness extending over the facial musculature. She says the movements of her lips gradually became limited until she could no longer co-apt them as in the act of whistling. The strabismus developed in both eyes, and the movements of the eyes became limited in all directions. Since this defect of the eyes there gradually developed a continuous lachrymation. She says she has decreased in weight from 153 to 106 pounds.

Examination.—On close examination we found her eyeballs fixed, there being no movement in any direction. They were also slightly protrusive. Excessive lachrymation was constant during the waking hours. The pupils reacted to light and in accommodation, and ptosis of left eye was marked; slightly so in the right. The diplopia, formerly so annoying, is now only present when certain lateral head movements are made since ophthalmoplegia has supervened.

Face.—The whole facial expression suggests a putty-like blankness. The muscular tonicity is lost, so that a state of profound paresis on the right side exists with a less degree of paresis on the left. The lips cannot be co-apted sufficiently to produce labial sounds, neither can the patient whistle. Attempts to show the teeth or to produce laughing expression distort the face slightly to the left. The masseters are about equally parietic and it is impossible for the superior and inferior maxillæ to be approximated with any degree of force. The chewing of bulky and solid substances cannot be done for want of power. There is no atrophy, tremor or fibrillary twitchings of the muscles involved.

Organs of Speech and Deglutition.—The tongue is thick and flabby, without indentures, atrophy or tremor, and is utterly devoid of any lateral movements. It can only be protruded to the inner margin of the lips. The palate has lost its symmetrically arched outline, and hangs low, with the uvula directly on the base of the tongue, and does not rise to close off the posterior nares in attempts to say "Oh," etc. The voice is exceedingly thick, and all sounds are indistinct, and finished off with the cleft-palate nasal twang. Her attempts to answer the necessary questions in the examination cause her much fatigue. The constrictor muscles of the throat are quite parietic, and deglutition of solids is difficult, while liquids regurgitate through the nose. A careful examination of the organs of voice by Dr. Hal Foster revealed paralysis of the left vocal cord.

Trunk and Extremities.—She is thin, as would be expected when remembering she is but a medium-sized woman, having

lost 47 pounds in weight. There is no atrophy of shoulder, arm, forearm, hands or trunk muscles, neither are there any fibrillary twitchings to be seen anywhere on close and repeated examinations. The power in all the extremities seems normal to the usual tests for one in her physical health, and there are no sensory symptoms present.

Reflexes.—The superficial reflexes are normal, whilst the deep reflexes (knee-jerks) are slightly exaggerated. No ankle-clonus.

The electric irritability of muscles is normal, and there is no evidence of disease of the heart or lungs. Urinary analysis proved negative. The pulse was 90, and regular. Menses and bowels were also regular. Appetite fair, notwithstanding the great loss in weight.

It would seem that while this patient was subject to remissions and exacerbations since the advent of her affliction, these variations have not been so markedly manifest as in the average case. When she did enjoy an apparent remission it was more of an arrest of the progress of the disease for a time only, to be broken by a sudden onward march to the decline. There appears to have been no special epochs when there was a cessation of her symptoms, with pronounced steps toward recovery, as was noted in Collins' report. It is true her amyosthenic manifestations were aggravated more at times than at others, but these were really brought on by slight efforts causing fatigue, and were largely corrected by removing the fatigue. The knee-jerks in this case while absent on the first test were later exaggerated, but grew less responsive on repeated tests, however, until only a minimum reflex act could be obtained. They were also restored to former activity by a period of rest. Any attempt to tax any organ or organs was soon followed by distressing fatigue and general nervousness. Severe attacks of tachycardia with prostration and weakness were sure to follow over-expenditure of her limited strength; but that there was actual improvement at any of the seeming periods of better health over and above arrested progress, there is no evidence.

A symptom attracting attention was the profuse lachrymation, a symptom common in true bulbar palsy, but usually attended by more or less emotion, which was wanting in this case. She was perfectly clear and responsive mentally, and remarkable for her buoyancy and composure. It must be re-

membered that complete ophthalmoplegia existed, and while ptosis was marked, there was a protrusion, seemingly of a naturally large eyeball, which remained unprotected from the particles of atmospheric dust ordinarily whipped away by the physiologic activity of the eye lashes. Exact observations on the working of the vocal cords are largely wanting in the literature on this subject; the nearest expression is the supposition of a paretic condition of the vocal cords, such as mentioned in Hoppe's case. Of course, paralysis of the palate, tongue and lips is disastrous to perfect externalization of speech, but the case presents the additional defect of paralysis of the left vocal cord. To be positive of this the competent counsel of Dr. Hal Foster was asked. In attempts at phonation the left vocal cord refused to functionate, was relaxed, passive, and did not rise to meet its fellow of the opposite side in the median line, and presented a pale anemic appearance. The right vocal cord, on the other hand, presented no abnormal appearance, and in functioning passed well across the median line in an attempt to approximate its derelict companion.

At a glance it will be seen that the symptom-complex making up the clinical picture is purely a motor defect. It is a typical denotation of suspended motorial function. As already mentioned, no evidence of superficial or deep sensory defects were manifest. The complete ophthalmoplegia externa (there being normal response of the pupil to light reflex and in accommodation) points to involvement of the third and sixth nuclei; bilateral weakness of the muscles of mastication sufficient to prevent the chewing of solid substances implicates the motor branch of the fifth; the facial palsy on one side and distinct paresis on the other, giving unilateral facial deflection on muscle functionation, and the blank non-expressive appearance in a state of quietude, is due to suspended innervation from the seventh nuclei; and a paralytic state sufficient to prevent phonation, vocalization, articulation and deglutition points to the 9th and 10th nerves, whose motor fibers arise in common from the nucleus ambiguus, which is a continuation of the motor cell-column of the anterior horn, while the 12th nerve is responsible for the lingual disability.

Diagnosis.—In the diagnosis of asthenic bulbar paralysis we are called upon to differentiate more particularly from

pseudo-bulbar paralysis and true degenerative bulbar paralysis. From the former, as Dr. Collins says, "it must always remain a difficult question, as in both there is absence of atrophy in the paretic muscles and in both the symptom-complex may be only atypically that of true bulbar paralysis."

Mills⁴ calls attention to the fact that in the majority of cases of pseudo-bulbar paralysis reported, the patients were aged and their vessels highly atheromatous. He also states that "one of the most important points of distinction between true bulbar paralysis and pseudo-bulbar paralysis is the condition of the tongue, which in the former becomes markedly atrophied and is the seat of fibrillary contractions from which in the latter it is free."

While it is true that a striking feature in our case was the absence of atrophy in any of the affected muscles, yet there was evidence of paresis of every motor cranial nerve beginning with the third and ending with the twelfth. The face, shoulders and limbs were remarkably well rounded, considering that the patient had lost 47 pounds in weight. The muscles supplied by the facial, motor fifth, and third nerves were, however, markedly affected, but the effect of the paresis was somewhat transient in character, the patient partially recovering the normal use of these muscles between the paroxysms, which according to Mills is the rule in the asthenic type.

In genuine bulbar paralysis the hypoglossus, vagus and glossopharyngeus are more seriously and continuously affected. No positive evidence of fibrillary twitchings could be elicited as being present, or as having been present. The reflexes were retained with the knee-jerks plus. The electrical response was normal. The psychical faculties were in no wise affected. The ciliary muscles were not involved, and the vision was normal. There was no dribbling of saliva. The tendency to fatigue and prostration was even more manifest on the slightest exertion, especially in muscles supplied by the motor cranial nerves, while to some degree the whole organism suffered. There were no disorders of common sensibility or special senses. Thus it would seem that the case is not one of glosso-labio-laryngo-pharyngeal paralysis, but is the clinical picture of a genuine case of asthenic bulbar paralysis.

⁴ Mills, Text-book on Nervous Diseases.

DISCUSSION.

Dr. Joseph Collins thought that asthenic bulbar paralysis was a sufficiently rare disease to warrant the recording of a new case. The case that Dr. Sinkler reported seemed to parallel the one published by Dr. Collins, except in one or two particulars. Dr. Collins did not believe that the exhaustibility of the muscles by the faradic current, as pointed out by Jolly, would be corroborated by future investigations. In Dr. Collins' case the muscles were exhaustible on certain days, but on other days no more exhaustible by the faradic than by the galvanic current, though more so than in the normal individual. Then the knee-jerks were continually present in Dr. Sinkler's case, but in Dr. Collins' case they were present when the patient was comparatively well; absent when she had bad days, and exhaustible at all times; *i. e.*, they became less vigorous under repeated blows and after the seventh, eighth or ninth tap were entirely exhausted.

Dr. Collins was not inclined to look upon the disease as one of the motor nerves primarily. It seemed to him that there is very little in the clinical manifestations to warrant us in taking the disease out of the domain of the sympathetic nervous system. True, its phenomena are manifested largely through the motor system, but its most important symptomatology is due to trouble in the sympathetic system. That had been firmly impressed upon Dr. Collins in the observation of his own case. He sometimes found his patient in a state of collapse, with ballooned abdomen, blue lips and blue finger tips, cold extremities and feeble pulse; and if it were not for the subcutaneous injection of large amounts of normal salt solution and the use of diffusible stimulants, he was sure she would have died just as patients do from surgical shock.

The most plausible theory of the pathogenesis of the disease, it seemed to him, was that in all probability there is some poison or infectious material generated within the system, continuously operative upon the vegetative portion of the body. This toxic material manifests itself periodically, and whenever the vitality is sufficiently low to allow the poison to get the upper hand, the phenomena of relapse appear. What the source of such a poison is, we can only conjecture. The point brought out by Dr. Sinkler, that there was a weakness of the nervous system manifesting itself in other members of the family, would only go to show that this disability may be in part inherited as well as acquired.

The remarks that Dr. Sinkler made concerning the necessity for differentiating the disease from hysteria seemed to Dr. Collins to be very well taken. The case reported by Dr.

Collins had been treated by specialists and general practitioners as one of hysteria.

A symptom that had not been mentioned in the papers read was one in relation to the menstruation. Dr. Collins' patient was always worse at the menstrual periods. She had great menorrhagia and on two occasions he had to pack the uterus to stop the hemorrhage.

Another very remarkable symptom or group of symptoms in his patient occurred in the shape of an acute psychosis, taking the form of confusional insanity. Any attempt to explain the occurrence of this must be in line with the statement that the poison which is in the system accumulates and at the time of the paroxysm exercises its effect upon the cortical cells as well as upon the other cells of the central nervous system.

Dr. W. G. Spiller referred to the fact that Dr. Sinkler had laid especial stress on a symptom that is very important in asthenic bulbar paralysis—the symptom of fatigue. In Dr. Punton's case restoration of function after rest was not nearly so prominent and Dr. Spiller was not at all certain that Dr. Punton's case was not one of pseudo-bulbar paralysis or possibly one of "bulbar paralysis without anatomical findings." The case, however, might be one of asthenic bulbar paralysis. The bulbar diseases still need much study.

Dr. Spiller said that a necropsy had been obtained in a case of asthenic bulbar paralysis reported by Widal and Marinesco, and changes in the nerve cells had been observed.

Dr. H. M. Thomas said he had had a case under observation for two years that seemed to him to be one of asthenic bulbar paralysis in the early stages. No permanent paralysis of the motor nerves of the eye or of the muscles of the face was present. The patient was in pretty good condition on getting up in the morning. She could speak fairly well for about half an hour, and then her voice gave out. When she began to eat her breakfast, she ate well enough until she had masticated for a few minutes, and then it became progressively harder for her to chew. If she was required to look up more than four or five times in succession, ptosis became evident and weakness of the superior rectus of each eye developed. Unfortunately the patient lived in another city and he had not been able to see her for a year, but had heard that she had periods of improvement and relapse.

Dr. Thomas had also had under observation another case that puzzled him a great deal as to diagnosis. The patient was a young girl who had the symptom of fatigue well marked, more particularly in the muscles of the extremities than in the muscles supplied by the cerebral nerves. She had never had a

complete remission, but was always better in the morning before she took any exercise, and got progressively worse towards evening. Dr. Thomas believed that the case was not one of muscular dystrophy.

Dr. Sinkler did not wish to go into a discussion of the pathology of the disease, as it was too vague and uncertain. He only wanted to refer to two symptoms in his case that he failed to emphasize sufficiently, namely, the absence of any muscular tremor even in the tongue, and the absence of atrophy.

Dr. Punton said that the question of syphilis was very carefully considered in his case and also the question of pseudo-bulbar paralysis, and they had come to the conclusion that the case did not belong to either class, and that it was one of asthenic bulbar paralysis.

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173. A PROPOS DE LA CHORÉE VARIABLE DE BRISSAUD. TROIS OBSERVATIONS DE CHORÉE (On Brissaud's Polymorphous Chorea. Three Observations on Chorea). A. Couvelaire et O. Crouzon (*Revue neurologique*, 7, 1899, p. 399).

Brissaud in 1896 described a new type of chorea, differing from that of Sydenham and of Huntington, which he called the "chorée variable des dégénérés." This new type, which may be termed "polymorphous chorea of degenerates," is characterized by the multiplicity and variety of its movements, by its remissions and variability in time, and also by the presence of various stigmata of degeneration in the subject affected. The authors present three cases from Marie's clinic, at the Bicêtre, illustrative of the condition. The first occurred in an epileptic. Her movements were bizarre and at times extremely violent, not permitting her to hold any articles in her hand. She had attacks of petit mal. The chorea was cured. The second case is termed a chronic non-progressive chorea. In this case the movements were greatly accentuated, though at first sight no marked abnormality could be detected. On close scrutiny, however, quick movements of the hands, lips, fingers, shoulders, eyelids, could be detected. At times the patient could not sit quiet. There were no degenerative stigmata in this case. The third case is one of irregular Sydenham's chorea. The second case more closely corresponds to Brissaud's type, though it lacked the stigmata. JELLIFFE.

REPORT OF A CASE OF TRIGEMINAL PARALYSIS.¹

By SANGER BROWN, M.D., Chicago.

PROF. OF DISEASES OF THE NERVOUS SYSTEM, POST-GRAD. MED. SCHOOL;
ATTENDING PHYSICIAN FOR DISEASES OF THE NERVOUS SYSTEM
TO THE ST. ELIZABETH'S AND ST. LUKE'S HOSPITALS.

The successful excision of the Gasserian ganglion by surgical procedure has afforded an exceptionally favorable opportunity for the physiological study of the trigeminus. A perusal of the more recent literature on the subject, however, still discloses many wide differences of opinion among students and investigators regarding the functions of this nerve. It is, therefore, desirable that cases in which the nerve has become paralyzed by the influence of some pathological process, as well as those in which various of its branches have been divided surgically, or the Gasserian ganglion itself removed, should be carefully studied and reported, so that from the mass of evidence thus accumulated conflicting views may be finally harmonized. Cases of trigeminal paralysis have, then, a double interest, clinical and physiological.

After all, it is not an easy matter to draw exact conclusions either from clinical or operative cases, if I may be allowed to make that distinction, for in the former it is often impossible to determine the exact limits of the influence of the pathological process producing the symptoms, and in the latter, on account of the surgical inaccessibility of the ganglion, more or less injury to contiguous structures can hardly be avoided. Then, too, the question being one which relates mainly to the various forms of sensation, the personal equation has very often to be reckoned with, which, of course, properly warrants more or less skepticism of published results.

The wide variation, not to say contradiction, which patients make from time to time in reference to their sensations might tempt the reporter to omit or suppress certain statements in order that his report might sound more reasonable or read better, but this would greatly vitiate its value, because, as already implied, activity of the pathological process, susceptibility of the nervous tissues, mental and emotional states, each and all

¹ Read before the Chicago Neurological Society, Feb. 23, 1899.

are vital elements or factors in the problem, and normally subject to very considerable fluctuation. A fair understanding of the personal traits of the individual upon whom the tests are made, however, is essential to the most correct estimation of their value, and therefore I shall now state that my patient is a well educated, cultured gentleman, who has always been conspicuously neat in his dress and personal habits, and thus might be expected to give more than ordinary attention to any deviation from the normal in any of his sensations. Indeed, what with the explanations and directions I gave him, and his natural tendencies, he very soon became a deeply interested and intelligent student of his own case, as might be surmised from his own written report of his symptoms, which I shall presently quote.

Mr. A. B., druggist, consulted me January 21, 1896; aged 57; married; lues denied; family history good, and personal habits irreproachable. Has always been an active worker, and enjoyed excellent general health. Had accumulated a handsome fortune by his own efforts, when a few years ago some of his property began to depreciate through causes which he could not foresee or remedy, and this in connection with some domestic trouble, has kept him constantly under a very distressing state of mental tension for some time. He had some irregularity of the heart six years ago, but recovered under the Schott treatment. Five years ago he had a severe attack of sciatica on left side, resulting in peroneal paralysis, with anesthesia of dorsum of foot, for which I treated him with electricity for about four weeks, when he recovered with the exception of some reduction of sensibility on the dorsum of the toes, which has persisted ever since. Otherwise his health has been excellent.

About two weeks ago, when brushing his teeth, he noticed a reduction of sensation in the gums of the upper maxilla of the left side, and a few days later lack of feeling in the upper lip of that side, and finally over the whole side of the face, including the scalp up to the middle line and backward nearly as far as the ears. He has noticed for the past day or two that the cheek was slightly swollen and the eyelids slightly stiff. Has not had a particle of pain. Got an eye-winker in the left eye yesterday and felt it as usual (?); has not noticed any defect in taste, or difficulty of mastication.

Examination. Sensation to pain and touch distinctly reduced throughout distribution of first and second branches of

fifth nerve. The reduction is greatest on cornea, gums, mucous membrane of cheek, and a circular area about the size of a silver fifty cent piece a little below the eye. Elsewhere sensation is normal, except a very slight anesthesia of the dorsal surface of the toes of the left foot, resulting from sciatica five years ago, as stated. Skin and tendon reflexes normal; no ataxia nor swaying with eyes closed; taste on the anterior two-thirds of tongue, left side, seemed reduced for bitter. The muscles of mastication appear to contract equally on the two sides. The pupils are equal and react normally to light and accommodation. Vision was not tested, but patient stated that he had occasion to use the eyes a great deal and had noticed no defect.

Treatment. Grains five iodide of potassium after meals; grain 1-32 strychnia before meals; galvanism 8-10 milliamperes to face ten minutes, three times a week.

January 28. Anesthesia has increased in the region of the second branch, and patient thinks decreased in first.

Smell has been repeatedly tested, and does not appear to be much, if at all, affected, but the septum on the left side appears to be entirely anesthetic. Two days ago patient had symptoms of a general cold, including sneezing, and took ten grains of Dover's powder; next day had pain for several hours, not very intense, in afflicted parts. Yesterday noticed difficulty in mastication on account of weakness of the muscles on left side, and found himself, contrary to his habit, using the other side. General health and spirits excellent; no pain except "soreness" of left side of tongue. A few days ago there was slight edema of left lids; none at present. No lowering of sensibility could be demonstrated in the region supplied by third branch to-day.

February 7. Began to notice that the lower gums on the left side were insensitive and anesthesia more pronounced in other parts. For several days has been taking 3 iii syrup hydriodic acid *t.d.* instead of 10 grains of potassium iodide. He was put back on iodide of potassium; suffers with insomnia, but has no pain. Muscles of mastication on left side still get unduly tired.

February 10. Upper and lower gums quite insensitive, and though there is hyperesthesia to the galvanic current over the left side of the face, sensation to touch and pin-pricks is distinctly lowered. The cornea can be touched firmly with the end of a pen stalk without producing any pain or reflex whatever.

February 12. Patient distinctly better for the past two days; less anesthesia of all parts except the upper lip and gums.

February 14. A spot about the size of a half dollar in the

center of the cheek below the eye is very hyperesthetic to the galvanic current and to pin-pricks, but when pressed with the finger feels as if there was a thickness of cloth—about medium weight broadcloth—between the finger and the cheek. The left side of the tongue is now hypersensitive to touch and taste in its anterior two-thirds. The gums react to the galvanic current and to pin-pricks and touch about the same on the two sides, but the cornea is quite insensitive to touch.

February 24. For the past three days patient has complained of marked numbness of the whole area of the fifth. While he feels a touch and prick in most places, it does not feel natural; feels as it might through a thin glove. He states that the muscles of mastication are growing distinctly stronger; do not tire as before. Corneal reflex still absent and cornea insensitive.

February 28. Patient has suffered for one week with dimness of vision, but when the eyes were tested separately with usual glasses vision was found to be 20/30 in each eye. Last evening, however, he developed marked diplopia, which appears to be due to weakness of the left external rectus. His subjective sensory symptoms are much less than two days ago. Referred to Dr. Henry Gradle for visual complications. (See Dr. Gradle's report below.)

March 6th. One week ago iodide of potassium was increased to gr. 20, *t.d.* For last three days face has felt almost normal, but diplopia constantly marked.

March 9th. Paresthesia somewhat worse in second branch, especially on cheek. Diplopia rather more marked. With my consent patient discontinued all medication and took a trip to California.

I met the patient casually several times between the period of his return from California a few months subsequent to the last entry and February of last year (1898). He assured me that there had been no material change in his symptoms, excepting that his diplopia had been much improved by an operation performed by Dr. Gradle; that his financial reverses had grown steadily worse, and he had exerted himself to the utmost to arrest them; yet his general health and capacity for work were excellent. At my request he readily agreed to take careful note of his symptoms covering a period of several months and write out the result for me. He gave me the following report about three months ago:

"I have been watching the conditions carefully, but have refrained from making any notes on account of the surprising changes constantly taking place. The variations are slight. Frequently of the numbness there is very little for a time, then

it is more apparent. These changes are of frequent occurrence, but on the whole the anesthesia is gradually lessening, it seems to me, possibly because I am becoming accustomed to the condition. In certain parts of "b" [Fig. 1] the sensations are often peculiar. I have felt a trickling as of cold water down that locality, but on putting my hand there I have always found the part perfectly dry and not even chilly. Again, a flash of heat will be the sensation when the fingers tell me there is no elevation of temperature; a sensation as of soreness.

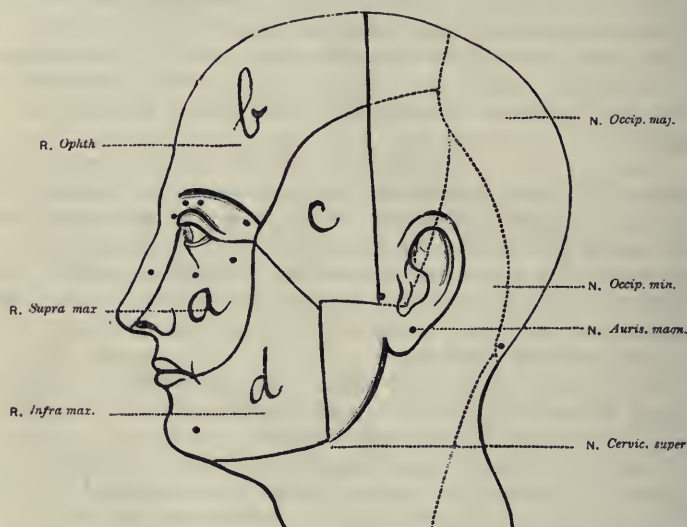


Fig. 1. At a, especially in central part and over cornea, the anesthesia is greater and more constant than elsewhere, excepting the external surface of upper gums and the contiguous mucous membrane of the cheek.

occurs, yet the part is not sore to the touch. At "c" [Fig. 1] the numbness seems to remain about constant, gives me no annoyance whatever; at "d" [Fig. 1] the anesthesia is at times very slight and never intense. The upper and lower lips on left side vary intensely from nearly normal to absolute anesthesia. Mastication is entirely on the right side, because I am liable to bite the left cheek and lips if performed on the left side. An almost invariable sensation of stiffness and swelling results at times of or after eating, and this seems particularly aggravated if salt and pepper, or either, comes in contact with the parts. Yet I find no difficulty in using the muscles and moving the parts, nor is there any distention that I can discover. This sensation does not last long at any time, yet is

very unpleasant. A sharp, stinging sensation occasionally darts through the left eyeball. It is not absolutely painful, feeling as though a fine cambric needle had been sent through it with the rapidity of lightning, and the sensation is gone. The teeth and upper and lower jaws on the left side are absolutely without sensation, except at times when a shock as of an electric current passes, and I can repeat this by bringing the teeth in contact. This sensation is occasional, not constant. An itching sensation akin to pain is not infrequent in the left eyebrow; it is not painful, and rubbing for a minute or two with the thumb dissipates it for the time. The left eyeball is absolutely without sensation at any time, except as before stated. Health perfect; digestion good; able to do any amount of mental or physical work; eat fairly well, but am never hungry. Sense of taste on left side of tongue varies; sugar is most readily detected, because most readily soluble; salt next; strychnia next; flavors are detected, but less readily than on the other side. I find it impossible to sleep more than two to four hours unless I take 10 grains of sulphonal on retiring, when I can readily sleep five to six hours. When not particularly troubled or anxious sulphonal is omitted three or four nights in succession, and sleep is natural during that time for from four to six hours. After lunch each day I sleep readily for twenty minutes, but omit this without inconvenience if I am occupied or have appointments to keep. All heart disturbance has disappeared. Vision is good in both eyes, but left eye does not focus with right, and there is a blurring sensation in consequence. Roof of mouth on left side is without much sensation, either to touch or taste. Left side of nose inside at times nearly normal; at others almost insensible to touch. Exposure to cold air causes a sensation of pain and stiffness on left side of face, which is very uncomfortable; yet there is no pain or stiffness when the parts are pressed or manipulated. Left side of tongue has a nearly continuous sense akin to soreness, the cause of which I am unable to determine. It may result from contact with the teeth or with food, or both; the numbness preventing the cause of the irritation being noticed at the time of its occurrence. Occasionally particles of food are swallowed which have not been sufficiently masticated, the effort to swallow giving the only intimation of the fact.

"The foregoing is a very brief summary. Much more could be noted had I the opportunity to describe the peculiar sensations at the time of occurrence, and some of the sensations would be difficult to describe under any circumstances."

February 10th. Patient re-examined to-day. There is marked ptosis of left side, but the ocular condition will be much

better described by Dr. Gradle than I could describe it. Extensive, if not complete atrophy of the temporal muscle is evident on inspection, and the same would probably be true of the masseter were it not obscured by the beard. When the patient bites only a feeble contraction can be felt. The lower jaw cannot be moved horizontally to the right beyond the upper, showing that the action of the left external pterygoid is deficient. The palatal arches are equal in repose, in movement, and sensation. Taste, touch, and the pain sense are normal on the posterior third of the tongue. Other conditions, exclusive of those described in Dr. Gradle's report, remain the same as three years ago.

Calling to mind the anatomy of the parts, it may readily be understood how a superficial neoplasm or aneurism at the side of the pons, developing quite rapidly at first and afterwards very slowly, might account for the symptoms. Tabetic changes affecting the nuclei of the several nerves, or the fibers emanating therefrom, might suggest themselves. A deep neoplasm involving the nuclei and fibers may, I think, for obvious reasons be excluded, and so also at this time may a superficial neoplasm of malignant type. Neither is it hardly conceivable that a syphiloma should enlarge so rapidly at first and afterwards so slowly, and that there should have appeared no other evidence of lues in the meantime. An aneurism might enlarge quite rapidly at first and then very slowly, corresponding to the successive invasion of contiguous nerve trunks. The age of the patient, too, is favorable to aneurism. Its presence, however, might be expected to injure to some extent the dura and adjacent bone on the one hand, and the superficial transverse fibers of the pons on the other; thus, giving rise to pain, and also symptoms of pressure upon the pyramidal fibers passing through the pons, both of which were absent.

The sensory symptoms of tabes, even when the disease commences in the brain stem, are usually bilateral nearly from the first, and almost never remain so strictly unilateral and so limited for so long a time as they have been observed to do in this case. Neither do they develop so rapidly at first and then slowly and gradually, involving consecutively nerves of which the trunks are contiguous, but the nuclei far apart and separated by other structures, the invasion of which must cause symptoms here absent.

Transient and recurrent diplopia is a common symptom of tabes in the earlier stages; but the same cannot be said of diplopia depending upon atrophy of ocular muscles; for assuming that a single lesion is producing the symptoms in this case, then, inasmuch as it has demonstrably caused destructive changes in the neurons composing the motor root of the fifth, it is not unfair to infer that the fourth, sixth and third have suffered in the same way. Again, it is highly improbable that early tabetic changes should show such a preference for motor structures as is here manifest; that is to say, having invaded four motor nerves and only one sensory nerve. Neither can the visual symptoms be regarded as affording any particular support to the diagnosis of tabes.

If one of the degenerative changes of the nervous system must be held responsible for the symptoms, then that pathological process which, when it attacks the peripheral motor neuron forms the basis of the clinical condition commonly designated progressive muscular atrophy of the spinal form, may be brought into requisition with far less distortion of its ordinary features than must be resorted to if tabes is employed for that purpose. Indeed, that form of progressive muscular atrophy known as bulbar paralysis occurs most commonly in people of about sixty, and though nearly always so is not invariably bilateral. I reported a case at the Mississippi Valley Medical Association, held in Louisville, September, 1897, in which the disease first appeared in the nucleus of the third nerve on one side, and in about eight months had nearly destroyed it, whereupon it appeared on the opposite side and advanced to complete destruction of both nuclei in about two years from its commencement. Soon after this it made its appearance in the cervical enlargement, as shown by atrophy of the thenar muscles of one hand, and a few months later those of the other, both hands becoming powerless less than two years later.

Finally, I repeat that, while not unmindful of the aberrant tendencies of so-called degenerative processes occurring in nervous structures, and those of tabes in particular, aneurism appears to me the most probable diagnosis.

If the disturbance of taste in the anterior two-thirds of the

tongue is greater than can be accounted for by the mere alteration of the tactile and pain senses then, of course, the case supports the contention of those who maintain that gustatory fibers from this part of the organ of taste pass to the brain through the trigeminus; but even so, no indication is afforded as to whether the course is through the otic or through the sphenopalatine ganglion. On the other hand, if the proper sense of taste is not lowered, then confirmation is afforded to the doctrine of those who contend that the pathway in question between the geniculate ganglion and the brain is via the nerve of Jacobson to the petrous ganglion.

REPORT OF OCULAR CONDITION.

BY HENRY GRADLE, M.D.

PROFESSOR OF OPHTHALMOLOGY, NORTH-WESTERN UNIVERSITY MEDICAL SCHOOL.

The patient, through the courtesy of Dr. Sanger Brown, came to me on the 2d of March, 1896. As I had known him for some years, I can fully corroborate what Dr. Brown has said regarding his education, superior attainments, and ability as an independent observer. He came to me on account of diplopia due to paresis of the left external rectus. Abduction was not much diminished, but there was slight convergent strabismus on looking straight forward. Paralysis of the abducens or external rectus does not by itself give rise to strabismus when the eyes are directed straight ahead, except in individuals whose muscles are not evenly balanced, and in whom the internus had the excess of power before the paresis occurred. When I first saw him there was convergent strabismus of six degrees, measured by the prism when looking directly forward.

He gave a history of severe coryza early in January, about two months previously, which persisted on the left side more than on the right. At the time he called there was no longer any acute inflammatory condition in the nose, but there was a purulent secretion, rather scant, from the left side. The septum was deflected to the left. The secretion came from the posterior regions, and may have been due to the inflammation of the sphenoid sinus. Some treatment by irrigation was adopted, which diminished this secretion and it almost disappeared. I

lay stress upon this because it led me to a view of the etiology of the case which the subsequent history has not fully confirmed. I have seen a number of instances in which paralysis of one or the other ocular muscle seemed to be directly attributable to nasal suppuration. In these cases the nose was narrow on the affected side, and there was the history of recent suppuration from that side, possibly in the ethmoid or sphenoid cells, a diagnostic point which is often difficult to determine with certainty. All of them recovered.

There was one point in this case that made me reserve my prognosis: Almost as a rule I have found that when there is strabismus on looking straight ahead, the prognosis regarding the recovery from a paresis is not as good as when there is no squint when the paralyzed muscle is not in activity. As Dr. Brown was directing the treatment with iodides, I limited my own treatment to the nasal suppuration.

The gentleman went on a Western trip, was much benefited in health, and while in San Francisco underwent a couple of nasal operations, I should presume, however, on a false diagnosis. The left maxillary sinus was punctured, but without finding pus. He was somewhat benefited, but I should judge more from the change of climate than from any active steps taken. Referring briefly to the subsequent nasal history, I can only say that the left side still discharges at intervals a scant muco-purulent secretion, but not continuously, and he has not cared for further surgical treatment.

The convergent strabismus increased, although the power of the abducens muscle did not diminish. In June, 1896, the condition had become stationary for so many months that I deemed an operation advisable for the relief of the diplopia in the median line. Accordingly, I made advancement of the left external rectus. The operation was done under cocaine and scarcely felt. Cocaine does not prevent the pain entirely in these operations on the contents of the orbit, but the pain was very much less than is ordinarily felt by sensitive patients; but, nevertheless, he was not entirely free from pain. The result of the operation, corresponding to the average success in such cases, removed the diplopia on looking straight ahead, but, of course, could not remove it when looking towards the paretic

side. Ten days later I made a tenotomy of the internal rectus and established a satisfactory condition, which persisted from June, 1896, until late in the summer of 1898.

Now, let me refer briefly to the intra-ocular condition. He has a low degree of astigmatism and a small amount of myopia in both eyes. With full correction his vision was 20/20 in both eyes. When he returned in May, 1898, he complained again of diplopia, and now, on testing him, I found the diplopia was vertical and no longer horizontal, except when looking in the direction of the paretic externus. There was now incomplete paresis of the left superior oblique, the muscle innervated by the fourth nerve. At this time the ophthalmoscope gave a normal fundus, but his vision of the left eye was not perfect, as when corrected fully I could not raise it to more than 20/30. I did not see him very often until August, 1898, when he complained more of diplopia, which had increased both in a vertical and horizontal direction. The success of the operation on the muscles moving the eye horizontally had not been diminished in the course of time, but paresis of the superior oblique had increased. At this time vision could not be raised to over 20/35. I did not see him again until a few days ago. Now the vision has sunk to 20/40, the myopia has increased in the left eye about a half diopter, and the astigmatism seems to have disappeared entirely. The ophthalmoscope shows no lesion, and the field of vision for white and colors shows no anomaly. I cannot account for the reduced sight of the left eye; it is not due to a retrobulbar neuritis. It may be due to a perineuritis of the intracranial nerve between the chiasm and the optic canal, but such a diagnosis is largely hypothetical. There are no other evidences pointing to any lesion anterior to the chiasm. When he returned in August, 1898, he complained of micropsia, objects appearing a little smaller to him when seen with the left than with the right eye. This may have been due to an optic condition, as the myopia had changed slightly, or it may have been purely of psychic origin.

When he returned a few days ago the paresis of the superior oblique had apparently increased slightly. But there was now a slight amount of ptosis of the left eye. It is a question in my mind whether this is due to involvement of the third nerve or

not. The ptosis is not the form we usually see in interference with the third nerve; the upper lid droops more as he looks downward, but can be raised to the normal extent when he looks upward. It is the exact reverse of the Graefe sign in exophthalmic goiter and there seems to be no actual loss of strength, as he can raise the upper lid voluntarily, and does so involuntarily as he looks up. The vision has now fallen to 20/40, without a sufficient intra-ocular explanation. Colors are seen with normal saturation, the false image belonging to the left eye being less sharply defined, but the tint of its color is the same as in the right, which would seem to exclude incipient atrophy of the optic nerve. The diplopia has ceased to annoy him, probably both on account of the poorer sight of the left eye and the screening by the partial ptosis. I considered it best not to advise any muscle operation, which he would willingly undergo, as it would no longer be a benefit to him.

We have here a gradual encroachment from the fifth nerve, and at the same time the sixth to the fourth, and now possibly to the third. But the involvement of the latter nerve is uncertain, as no other fibers of the third are involved, the pupils being normal in both eyes, and all the other muscles normal as far as one can judge. The pupils are sluggish, but perhaps not more so than corresponds with his age.

The anesthesia of the conjunctiva and the lids is, perhaps, a little more absolute than originally. At the time of the operation in June and July, 1896, the anesthesia was not quite complete. The cutting part of the operation was scarcely felt, but the dragging of the sutures was a little unpleasant. The nose I have not examined carefully recently for anesthesia. On previous occasions it had never been entirely anesthetic; he had felt manipulations with the probe somewhat unpleasantly. There is no appearance of atrophy of the mucous membranes. The areas of sensation, I think, have been described by Dr. Brown. His hearing is normal as to range. There is no interference with any of the secretions.

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174. BEITRAG ZUR LEHRE VON DEN PATHOLOGISCHEN BEWUSSTSEIN-STÖRUNGEN (Contribution to Pathological Conscious States). Schultze (*Allg. Zeitschrift f. Psychiatrie*, 55, 1898-1899, p. 6).

Schultze describes three cases of what may be termed ambulatory automatism. These he considers comparatively rare conditions, for Germany. These patients had all taken somewhat extensive trips away from their homes, without any aim and seemingly motivated by unknown impulses. Amnesia was present in varying grades. In two cases degenerative factors were not particularly prominent; in a third case dipsomania was present with other well-marked degenerative characters. The author is inclined to class all of the cases as post-epileptic automatic states, though in the discussion following this paper the opinion was strongly expressed that the condition undoubtedly existed apart from the epileptic taint, in which latter class of cases amnesia, however, is apt to be more profound.

BENOIT.

Society Reports.

CHICAGO NEUROLOGICAL SOCIETY.

February 23, 1899.

The President, Dr. Richard Dewey, in the chair.

Dr. Sanger Brown read a paper with the title: "Report of a Case of Trigeminal Paralysis." (See p. 554.)

Dr. H. N. Moyer had observed a case very similar to the one reported by Dr. Brown. Twelve years ago he was consulted by a hostler for paresthesia of the face. He said his face felt as if it had been kicked by a horse the day before. Two or three years later he developed anesthesia of the face and loss of taste. The anesthesia finally became complete and sensation to temperature, touch and pain was absolutely gone. Nevertheless, if the skin was moved he knew that he was touched. This seems to show that there is a muscle-sense apart from the pain- and temperature-sense. Later the patient developed eye symptoms, partial ptosis and paresis of ocular muscles, pains developed, the knee-jerk disappeared, and he is now, twelve years after the first observation, in the Kankakee Hospital, a typical case of tabes.

Dr Moyer reported the case of a man who had complete anesthesia of the middle trunk of the fifth nerve with bilateral paresthesia for several years, but in this case there was no disturbance of taste. He had had various ocular palsies, but of late had lost the knee-jerk, had a good deal of swaying when his eyes were closed and lightning pains.

In a third case there was total anesthesia in the middle trunk of the fifth nerve on one side with no impairment of taste, but with ulceration and opacity of the cornea, loss of knee-jerk and swaying when the eyes were closed.

On grouping these three cases, considerable variation in their symptoms will be seen: One had trophic disturbance of the cornea, one had disturbance of the taste-sense, and the others had not. When these cases are taken in connection with Dr. Brown's case it will be seen that as far as the physiological part of the nerve of taste is concerned, these four cases throw very little light upon it, and it must be considered that the sense of taste differs in different individuals. Dr. Brown's case appeared to be one of tabes.

Dr. Brown stated that the knee-jerks were normal and that there was no ataxia.

Dr. Moyer believed that the knee-jerks would disappear later. In the case he had observed there was not a sign of tabes for the first six or eight years the patient was under observation. He considered the trouble to be not in the Gasserian ganglion, but back of it.

In reply to a question by Dr. Gradle, Dr. Moyer stated that tabes was likely to result at any age and was not excluded by advanced age; and in reply to an inquiry by Dr. Dewey, Dr. Moyer said that in the first of his cases a luetic history was doubtful, in the second lues was probable, and in the third nothing could be determined.

Dr. Sydney Kuh asked Dr. Brown whether the tests for the taste-sense were made by him or the patient. Dr. Brown replied that they were made as a rule by the patient, who was cautioned to keep his tongue out of his mouth and hold the substance on his tongue until he tasted it or felt sure that he was not going to taste it. Dr. Brown made some tests in his office by applying solutions on cotton to the tongue, being careful that there should be no opportunity for the solutions to spread to other parts.

Dr. Kuh thought that the cases reported had thrown some light upon the course of the fibers through which the sense of taste is conducted to the brain. In Dr. Brown's case the first and second branches were affected to a greater extent than the third; in two of Dr. Moyer's cases the first and second branches only were affected, and in one case all three branches. These observations are very similar to those published by Schmidt in the *Deutsche Zeitschrift für Nervenheilkunde*, 1895, and all taken together seem to show that the fibers of the taste sense should be looked for in the third, and not in the second branch of the fifth nerve. The few observations in which the glossopharyngeal nerve was affected at the base of the brain seem to demonstrate that not all the fibers for the sense of taste pass along that nerve, because there is no record of any cases in which there was absolute loss of the sense of taste excepting in the posterior part of the tongue. The age at which tabes appears depends in great measure upon the age at which syphilis is acquired. He had observed one case of a man 51 years old who had had symptoms of tabes for a few months only, but who had acquired syphilis at the age of 36. In another case, the symptoms of tabes developed after the age of 60. One case is recorded in which the first symptoms of tabes occurred at 70, and in this case syphilis was acquired late in life.

Dr. J. J. M. Angear believed that the facial as well as the fifth nerve must be involved in this case. He himself had had paralysis of the facial nerve a few years ago and suffered for a few days from bad taste, although there was no difficulty with the fifth nerve. The origin of the fifth nerve is so extensive that part of it goes to the lower part of the cervical portion of the cord, and a part goes up to the fourth ventricle, the aqueduct of Sylvius, etc. The nuclei from the third, fourth and fifth as well as the facial nerve are very near each other, so that what interferes with the function of one may affect the functions of one or all the others.

Dr. Henry Gradle, having observed the case, remarked first, that there was no evidence whatever of involvement of the facial nerve. Second, that as the anesthesia came on gradually, has never been complete, and as the nature and seat of the lesion is unknown, we are not justified in drawing inferences regarding the paths of the gustatory fibers. Third, the cornea was normal. He also wished to ask whether the tabetic ocular paralyses were not more complete and more fugitive than in this case. Furthermore, the impairment of sight was slight, had taken a long time to develop, and was therefore not indicative of tabes. In tabes also the loss of vision is due to visible atrophy, and this was not the case in the present observation. He could not explain the amblyopia, as there was no interference with the fields for white or colors, nor with the central perception of color. He thought it improbable that a syphilitic lesion had existed for four years, encroaching gradually from the fifth and sixth to the fourth and third nerves without causing more acute symptoms.

Dr. Hugh T. Patrick expressed the opinion that although a large proportion of the ocular paralyses of tabes are comparatively transient, and although in the permanent cases a large proportion of the patients have a high degree of paralysis with marked strabismus, yet the ocular pareses in this case were not incompatible with a diagnosis of tabes. The optic atrophy was also a somewhat anomalous condition, but there may be a stage of the disease when there is some visual defect from optic atrophy, and yet the latter be so slight that the conclusion should be drawn from the condition of vision rather than from the appearance of the disk. The atrophy might affect the central more than the other fibers, although in that case the central perception of color should be

affected. He inclined to Dr. Moyer's view of the pathology of the case, that it was degenerative rather than inflammatory or neoplastic. He believed that he had seen an analogue of Moyer's first case in a patient sent to him by a laryngologist. Although a well defined anesthesia of the mucous membrane of the nose and the upper lip, where she complained of discomfort, could not be made out, it seemed to him that these branches of the fifth were involved. Examination revealed to him loss of the knee-jerk and a beginning Argyll-Robertson pupil.

Dr. Henry M. Lyman was reminded by this case of another in which there was atrophy of both optic nerves, Argyll-Robertson pupils, progressive deafness of both ears, with retention of the knee-jerks. This case seemed to be one of degenerative sclerosis involving the optic nerve and the ganglia at the base of the brain and progressing downward, the spinal cord not having yet become involved. In Dr. Brown's case the most probable hypothesis is a localized degenerative change at the point which impinges upon the roots of the nerves mentioned. Is the case tabetic in origin, or may it not be a case of progressing multiple sclerosis?

Dr. Moyer remarked in connection with the suggested diagnosis of tabes, that the bulbar forms of tabes, although exceptional, bear a strong resemblance to the ordinary forms of tabes beginning in the spinal cord. The process is on a level with the head instead of the trunk.

Dr. Brown, in closing the discussion, stated that the neurologist could fasten almost anything upon tabes, and tabes upon almost any class of symptoms. The advocates of tabes agreed that the ocular symptoms in this case were very unusual, and that the beginning unilateral sensory symptoms so strictly confined to one nerve trunk were not likely to be tabetic. He was led to exclude tabes by the rather rapid unilateral onset, by its remaining restriction, and more than all, by the involvement of the motor branch. His view of the nature of the lesion was only a suggestion. The only external growth that could account for the symptoms would be an aneurism. The objection to this hypothesis was that the pressure would not be confined exclusively to these nerve trunks, but would produce symptoms from involvement of adjoining tissues. The hypothesis of sclerosis seemed to be hardly tenable, as sclerosis in the area involved would almost certainly involve other structures. Krause, in his monograph, refers to Dr. Schmidt's cases as the most instructive of any that have been reported. In one of his cases the ganglion was completely excised, and the woman thought that she had a better sense of taste than before. In the other cases the taste was more or less lost in the anterior two-thirds of the tongue. We must at present accept the conclusion that these fibers have most diffuse paths.

UNUSUAL CASES OF MYXEDEMA.

Dr. Sydney Fuh read a paper reporting two cases of this disease. Both patients were males, the one fifty-one, the other fifty-two years old. They presented a number of the classic symptoms of myxedema such as: Atrophy of the thyroid gland, weak and low pulse, subnormal temperature, dry cold and scaly skin, and loss of sexual power. In both cases the myxedematous infiltrations were of an unusual character. In the first it was found in the cervical region only, it was crossed

by a scar from an old operation and was uncommonly firm, resembling a fibromatous keloid in appearance and consistency. When the treatment was interrupted, which happened repeatedly, the swelling would promptly reappear. In the second case there was at the time of the first examination only a slight edema of the eyelids, but after a few days a typical myxedematous infiltration was found in the cervical region, which repeatedly vanished after a short existence and then, in spite of continual treatment, reappeared for a few days.

In the second case the attendant mental disturbance consisted in many delusions of a hypochondriacal nature, with pronounced indications of mental weakness. An acute mental disturbance characterized by numerous hallucinations of an unpleasant nature and great excitement was followed by a complete loss of energy, without any other signs of mental disease or weakness.

The first patient was treated with desiccated thyroids and he continues their use up to this time. He has been well for about four years. In the second case iodothyryn was used, and this seemed to have less disagreeable secondary effects than the powdered gland. He, too, continues the drug and has had no recurrence of the disease.

Dr. J. B. Herrick had had some experience with myxedema of children, but very little with this condition in adults. The deleterious effects of thyroid extract as brought out by Dr. Kuh and observed by Dr. Roos are due to contamination. The nearer the approach to pure iodothyryn, the nearer perfection is the effect of the drug. The early search for the myxedematous deposit in the supraclavicular space is of great importance. If Dr. Kuh's observations are corroborated, and if it is found there long before it is observed in the face and other parts of the body, it will prove a valuable aid in early diagnosis. The appearance and disappearance of the swelling before treatment would seem to show that the thyroid retained what might be called an intermittent function. In the case of a cretin who lives at a distance, Dr. Herrick had found that the dose could be very accurately controlled by noting the condition of the bowels. The child who had previously been badly constipated was given just enough thyroid extract to induce one or two bowel movements a day, and in this way the right dose was apparently given.

The President had noticed some unfavorable symptoms and complications following the use of Armour's thyroid preparation, but not in myxedema. In one case of climacteric insanity the pulse became very high for a couple of hours, reaching 160 or higher. The respiration was not correspondingly affected, the temperature was 100 degrees, and the patient seemed comfortable, or at least not seriously depressed. The tachycardia disappeared in about three hours. The treatment was discontinued for the time, but was taken up later on, and eventually a complete recovery from the mental symptoms was secured.

Dr. Kuh, in closing the discussion, said that he believed the best results could be obtained in myxedema by very gradually increasing the dose until the first symptoms of intoxication appeared. In the second case reported the dose was increased until the pulse became ab-

normally frequent, when the dose was slightly reduced. In the first case in which the tablets were used, gastrointestinal disturbance, diarrhea, gastric distress, anorexia and increased frequency of the pulse were the chief toxic effects. The dosage was thereupon reduced.

The best results cannot be obtained by small doses, but by remaining just below the toxic stage, until the symptoms disappear. Small doses should then be given every day or every third day. Unless the patients can be watched closely, they should be kept in bed while full doses are being given.

175. PARALYSIE GÉNÉRALE INFANTILE AYANT SIMULÉ L'IDIOTIE (Infantile General Paresis Simulating Idiocy). Toulouse et Marchand (Soc. méd. des. Hôpitaux, 1899, June 23).

The recognition that a form of dementia closely resembling general paresis may occur in the young is becoming more and more generally admitted, but, according to our authors, one point has not received sufficient attention. That is, that many cases of paresis occurring in the very young are often mistaken for cases of idiocy. In a case reported the progressive dementia commenced after a period of apparently normal development. There were inequality of pupils, speech disturbances, rapid emaciation, epileptiform attacks. The autopsy findings showed cerebral atrophy, adherent meninges, proliferation of glia, and other changes held to be typical of paresis. The child's father had died of paresis, with syphilis, and the possibility of chronic meningo-encephalitis is discussed by the writers. JELLIFFE.

176. UEBER SCHÄDELOPERATIONEN BEI EPILEPSIE (Trephining for Epilepsy). Matthiolius (Dent. Zeitsch. f. Chirurgie. 52, 1899, pp. 1 and 2).

That certain cases of traumatic Jacksonian epilepsy have been much improved is well authenticated, but the results in non-traumatic cases have not been as satisfactory. Four new cases are reported here. Two for non-traumatic epilepsy, both with negative results, and two of the traumatic Jacksonian type, with one recovery.

Matthiolius also presents the statistics of 164 cases that have been under observation, of which number 89 were non-traumatic partial epilepsies, 52 generalized traumatic types, and 23 of general idiopathic epilepsy. Of this total some 24 were healed; 25 improved; 31 died; the rest remained about the same. Thus there was 29.8 per cent. of favorable results, as against 70.2 per cent. failures. JELLIFFE.

Periscope.

PATHOLOGY.

177. UEBER DAS BLUT UND DESSEN BACTERICIDES VERHALTEN GEGEN STAPHYLOCOCCUS PYOGENES AUREUS BEI PROGRESSIVER PARALYSE (The Blood and its Reaction to Staphylococcus Pyogenes in General Paresis). H. Idelsohn (Arch. f. Psychiatrie, 31, 1898, p. 640).

The author gives here a short résumé of the work done on the morphology of the blood in this disease, and records the results of a series of experiments with some 38 cases, on the bactericidal activity with the staphylococcus pyogenes aureus. He contributes no observations bearing on the morphology of the blood, but concludes from his other investigations that whereas in the serum of normal individuals and in mental cases not afflicted with paresis, there is, to this bacterium, a distinct positive, bactericidal action, in general paresis, however, such action is not present. In a few cases there was observed a retardation of the growth which, however, never reached the point of a positive anti-bacterial action. This is explained on the ground that in these patients a certain amount of chloral had been administered. This loss of bactericidal activity is held by Idelsohn to be specific and does not depend on the condition of nutrition of the patient, though what is the cause is not brought out by the experiments in question. Changes in alkalinity, diminution of NaCl content, changes in leucocytes or modifications of alexins are all suggested as theoretical explanations. The author further thinks that this reaction can be used as a diagnostic factor in the early stages of the disease.

JELLIFFÈ.

178. THE PATHOLOGY OF THE BLOOD IN VARIOUS PSYCHOSES. V. V. Kroumbmiller (St. Petersburg, 1898) (Revue de Méd.).

The writer, in an inaugural thesis, presents studies on the changes in the leucocytes in a number of cases of mental disease. The blood of fifteen patients, suffering from melancholia and mania, of three idiots and of twelve epileptics was studied, all of these patients being under the same conditions of regimen and treatment. In all of them, excepting the epileptics, the blood was collected in the morning, two days in succession, at intervals of one week. In the case of the epileptics it was examined during an attack. Ouskov's and Sollarch's methods were used for the staining and mixing.

The author found that in melancholia there was an increase of leucocytes. Lymphocytes and adult leucocytes were diminished. Old leucocytes, especially the eosinophiles and disintegrated cells, were remarkably increased. In idiocy the total number of leucocytes was found to be increased, the adult and the older leucocytes being augmented at the expense of the lymphocytes; the leucocytosis of idiocy and melancholia thus resembling each other, save that the metamorphosis of leucocytes is more active in idiocy, more transitional, young forms being found. In mania there would seem to be a hypoleucocytosis. In epilepsy, at the time of an attack, the total quantity of leucocytes was increased, and this increase became less with every new attack. This increase was due to the relative and absolute increase of young and adult cells, while relatively the old cells diminish. The

author also found that in this disease the red cells were diminished, the specific gravity increased and the spleen became smaller. He also observed that closely following the attacks the leucocytes were not as numerous as one hour later. A few hours after the attack there was an absolute and relative increase in the eosinophiles and disintegrated cells. The author holds that there are alterations taking place in the spleen during the epileptic attack which account for these changes.

BENOIT.

179. BEITRÄGE ZUR LEHRE VON DEN AUS NERVENGEWEBE BESTEHENDEN GESCHWÜLSTEN (Contributions to the Study of Nervous Tissue Tumors). H. Haenel (Arch. f. Psychiatrie, 31, 1898, p. 491).

Haenel describes some anomalous tumors of the dura in a patient dying of a sarcoma of the white matter of the right hemisphere, that had undergone cystic degeneration. These two tumors were located on the dura near the superior orbital fissure.

One was a true myelo-neuroglioma, about 2 cm. in diameter, and consisted of strands of nerve fibers, running parallel, having large cells at their terminal filaments. These cells, from their general shape and size and appearance, strongly resembled the ganglion cells of a spinal ganglion, especially the Gasserian ganglion.

The other tumor resembled a fusiform cell sarcoma, soft in consistency, which was partly made up of fibers from the other tumor. Hemorrhagic foci about the lymph spaces and proliferation of the endothelium gave it the stamp of Ziegler's lymphendothelioma. There was also pachymeningitis interna, obliterating endarteritis, and fatty degeneration of the dura.

JELLIFFE.

180. ZUR ANATOMIE DER SEHNERVENATROPHIE BEI ERKRANKUNGEN DES CENTRALNERNVENSYSTEMS (On the Anatomy of Optic Nerve Atrophy in Diseases of the Central Nervous System). A. Elschnig. (Wiener klin. Wochenschrift, March, 1899, No. 11, p. 275).

The author presents the results of a study of the anatomic conditions underlying optic nerve atrophy in two cases, one of disseminated cerebro-spinal sclerosis and one of tabes. In the first-named condition he believes the atrophy to be the result of a peculiar interstitial inflammation which occurs absolutely at random in localized portions of the nerve and causes first a rapid destruction of the nerve-sheaths and fibers, and then an increased formation of connective tissue. This process he considers identical—so far as its inflammatory nature is concerned—with that which gives rise to the central sclerotic changes. In the tabetic case, Elschnig found complete atrophy of the nerve fibers and ganglion cell layer of the retina, diminution in volume of the entire nerve, increase of glial tissue in its intraocular portion, and in the remaining portion a partial disappearance of the nerve fibers, this more marked in the distal than in the proximal portion; further, a disappearance of the finer branches of the connective tissue septa which were themselves, both absolutely and relatively, somewhat thickened; thickening and sclerosis of the walls of the vessels.

From the fact that the anatomical changes were most marked in the retinal end of the nerve and diminished in intensity centrifugally, the conclusion is drawn that the process which leads to optic nerve atrophy in tabes is to be considered analogous in its primary topographical origin, and in its subsequent developmental direction with that which involves the other sensory neurons in this disease; in other words, that it is a peripherally originating centripetal process which is in nowise to be considered a pressure neuritis in the ordinary sense.

COURTNEY.

181. MITTHEILUNG ÜBER EINE BESONDERE VERÄNDERUNG DER NERVENFASERN DES RÜCKENMARKS WELCHE EINER KLINISCHEN BEDEUTUNG ENTBEHRT, NÄMLICH DIE VON MINNICH SOG. HYDROPISCHE VERÄNDERUNG. (Communication concerning a Peculiar Change in Nerve Fibers in the Spinal Cord Without Clinical Significance; the so-called Hydraptic Change of Minnich.) Karl Petré. (Deutsche Zeitschrift f. Nervenheilkunde. 15, 1898-1899, 1 and 2, p. 31.

Petré describes an interesting form of alteration of nervous tissue which he believes is the same as that seen by Minnich in different diseases, and described under the name of "hydropic swelling of the nerve fibers. Schaffer's observations in cases of general paralysis he regards as similar to his own, although Schaffer gave no details of the microscopic changes in the nerve fibers. Petré has seen the "hydropic swelling" in a case of tuberculosis without clinical evidences of spinal disease, in a case of abscess of the liver with the clinical signs of septicemia, and in a case of tabes incipiens. The altered areas in spinal cords hardened in Müller's fluid appear less deeply stained by the bichromate solution, but are not distinguishable by the Weigert hematoxylin method. The findings obtained by Petré were swollen medullary sheaths and swollen axis cylinders, axis cylinders bent so that two or three twists were seen in a transverse section of the cord, axis cylinders poorly stained in their central portion or appearing as granules, medullary sheaths staining with carmine in their inner portion, and glassy swollen neuroglia. These changes were seen in the posterior and lateral columns, and were not exactly the same in each case. The altered position of the cord did not stain differently from the normal by Marchi's method. Petré finds that the change is one of diffuse character and not systemic, and it probably occurs shortly before death; because the neuroglia is not proliferated, the glia cells are not more numerous, the vessels are normal, and the alteration of the nerve fibers is extensive and intense, although clinical signs have never been caused by this change. It is not purely a post-mortem condition, and though it may possibly develop after death, it is dependent on some alteration occurring during life. It must be distinguished from the true degeneration with the formation of sclerotic tissue seen in pernicious anemia. The recognition of the existence of "hydropic swelling" is very necessary, as in several instances it has been described as a true degeneration occurring during the lifetime of the individual and not merely at the close of life. SPILLER.

182. UN CAS DE PARALYSIE ASCENDANTE AIGUE (A Case of Acute Ascending Paralysis). H. Roger and O. Josué (La Presse médicale, 27 July, 1898, No. 62, p. 44.)

The patient, a man of 33, stone-cutter by occupation, first noticed a sensation of intense cold in the legs. Three days later, these were swollen and gradually lost power, compelling the patient to enter the hospital. At no time was there pain. Later there was some sore throat, and finally a severe attack of dyspnea, followed in the course of a few hours by death. His appearance resembled that of a patient suffering from severe infection. The voice was hoarse, deglutition was imperfect, both legs were in a state of flaccid paralysis, the knee-jerks and the tendon reflexes of the arm were absent. There was slight anesthesia of the legs, and tachycardia. The autopsy was performed thirty hours after death, and failed to reveal any gross lesions. Microscopical examination of the spinal cord showed extensive degenerative alterations in the cells of the anterior cornua, consisting of chromato-

lysis and dislocation or disappearance of the nucleus. The neuroglia cells were much more numerous than normal, particularly around the central canal and at the periphery of the gray matter. Cultures made from the blood during life gave a single micro-organism resembling the pneumococcus. Portions of the medulla oblongata were injected in the anterior eye of a rabbit which did not show subsequently any signs of hydrophobia, thus excluding paralysis due to this cause. Other rabbits were inoculated with the culture of the pneumococcus, and four out of five showed paralysis of the posterior limbs before death. The other died of purulent pericarditis. One mouse survived, but one died. The authors believe that their case represents a form of Landry's disease, produced in this instance by the pneumococcus, the particular variety present showing apparently a peculiar predilection for the spinal cord.

SAILER.

CLINICAL NEUROLOGY.

183. "EIN PATHOGNOMISCHES SYMPTOM DER FUNCTIONELLEN (HYSTERISCHEN) EXTREMITÄTENLÄHMUNG" (A Pathognomonic Symptom of Hysterical Paralysis of the Extremities). Von Hoesslin (Münchener medicin Wochenschrift, 1899, No. 10, p. 313).

The author claims to have discovered an absolutely diagnostic sign of functional paralysis of the limbs. His test is carried out as follows: He orders the patient to extend the affected limb while he (the operator) opposes a graduated resistance with his hand. The resistance is next suddenly removed. In every case of paralysis from organic lesion, the limb jerks suddenly in the direction of the intended movement. In functional paralysis on the contrary, the movement is suddenly arrested, and then after a short pause proceeds in the intended direction. This is due to what the author terms, the "paradoxical contraction of the antagonists." While a given movement is being performed, the condition of the antagonist muscles is carefully tested. If they are in a condition of strong contraction the paralysis is functional. He has found no exception to these rules in all the cases of paralysis of the extremities which he has examined during the past four years.

ALLEN.

184. A CASE OF SPASMODIC TORTICOLLIS ASSOCIATED WITH CHOREA. E. Noble Smith (The Clinical Journal, No. 341, p. 41; May 10, 1899).

The patient was a girl of sixteen years, and the so-called chorea was said to have antedated the torticollis by nearly eight years, but there is nothing in the text to show that the affection was really chorea. When first seen she was suffering from spasmodic torticollis, said to have been due to the action of both sterno-mastoids. An apparatus was applied which relieved pain and controlled the spasms to some extent, but the author found it necessary to operate, removing about one-half inch of the left spinal accessory nerve, producing complete paralysis of the sterno-mastoid and upper fibers of the trapezius. A year and a half later there was no spasm in the left mastoid, although the power of the muscle had returned, but it was now found necessary to resect portions of the second, third and fourth cervical nerves. Two years and a half later there was still considerable spasm said to have been limited to the right sterno-mastoid, so the right spinal accessory was resected. In spite of the imperfect results of previous operations, and in spite of the fact that two weeks after the last operation there were spasms in the left sterno-mastoid and left platysma, the author seems to think that there was "every prospect of a good recovery."

The optimism of the operator may further be seen in his statement that this was the nineteenth operation of this kind which he had performed for spasmodic torticollis and that the result had, in each case, been satisfactory, followed immediately by the statement that in one case the first operation relieved the symptoms for a time and that all subsequent operations were of little or no value.

When the author comes to treat of the pathology it is very evident that he has no proper conception of the nature of the affection and is entirely unacquainted with the instructive writings of Brissaud on the subject. Such papers as this do distinct harm by apparently making simple a very difficult subject, and by throwing a surgical glamour over an affection that is distinctly medical. PATRICK.

185. DIE SOGENANTEN AUSSEREN DEGENERATIONSZEICHEN BEI DER PROGRESSIVE PARALYSE DER MAENNER. (The Stigmata of Degeneration in General Paresis). P. Nacke. (Allg. Zeitschrift f. Psychiatrie. 55, 1899 pt. 5, p. 140.

In the *Neurologisches Centralblatt* for 1897 Nacke published a preliminary contribution on this subject. The above article is an enlarged and critical résumé of a large number of his personal observations. When compared with normal men, there is a large preponderance of the external degenerative stigmata in paresis, almost amounting, according to Nacke's figures, to double. He does not believe that syphilis is anything more than a predisposing cause, although from 60-75 per cent. of his cases had been infected. The author also contributes to the discussion of the change in type of this disease. He finds cases of prolonged dementia more common than supposed, and fewer acute exacerbations. Pathologically, meningeal hemorrhages are considered rare, and the frequently described thickening of the pia, ependymitis, general hydropsia and ventricular hydropsia are thought to be less frequent.

JELLIFFE.

186. UEBER CHRONISCHE STEIFIGKEIT DER WIRBELSÄULE (Concerning Chronic Rigidity of the Vertebral Column). Aug. Hoffmann. UEBER ANKYLOSIRENDE ENTZÜNDUNG DER WIRBELSÄULE UND DER GROSSEN EXTREMITÄTENGELLENKE (Concerning Ankylosing Inflammation of the Vertebral Column and of the Large Joints of the Extremities). v. Bechterew. NEUE BEOBSACHTUNGEN UND PATHOLOGISCH-ANATOMISCHE UNTERSUCHUNGEN ÜBER STEIFIGKEIT DER WIRBELSÄULE (New Observations and Pathological Anatomical Investigations on Rigidity of the Vertebral Column). v. Bechterew (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, Nos. 1 and 2 pp. 28, 37 and 45).

These three papers may be considered together.

Hoffmann reports the case of a man of thirty-eight years who presented great rigidity of the vertebral column, associated with muscular weakness and occasional pain in the spinal nerves. Crepitation of the joints of the extremities or exudation into these joints was not observed, but it seemed probable that a rheumatic change had occurred in the shoulder and hip articulations. Treatment directed toward the cure of a very extensive acne eruption, warm baths, sand baths, antirheumatic remedies, resulted in a complete restoration of function. The rigidity of the vertebral column was believed to be caused by toxins produced in the acne pustules.

v. Bechterew emphasizes the fact that the "rigidity of the vertebral column with kyphosis" described by him is not the same disease as that described by Strümpell and Marie, in which the large joints

of the extremities were involved. v. Bechterew reports two cases like those of Strümpell and Marie, in which ossification of the large joints and a rheumatic etiology were noted, and the progress of the disease was ascending, while kyphosis and root symptoms were unimportant or absent.

In the third paper a typical case of v. Bechterew's form is described. Kyphosis of the upper thoracic and lower cervical vertebræ, without prominence of individual vertebræ and without compensatory lordosis; half-flexed and prominent knees when the patient was in the standing position, sensitiveness of parts of the spinal column on percussion, almost complete immovability of the spinal column in backward and lateral movement, breathing almost entirely of the abdominal type, atrophy of the muscles about the scapula and in the upper extremities, exaggerated tendon reflexes of the lower limbs, were the chief features of the case. The joints of the limbs were not affected. Syphilis seemed to have etiologically importance.

v. Bechterew has obtained a necropsy in one of his cases of rigidity of the spine. He found that the bodies of the vertebræ in the upper thoracic region were in direct contact on their anterior surfaces. Degenerated roots, especially posterior ones, were found in the lower cervical and upper thoracic portions of the cord, and to a less extent in lower levels. The posterior and anterior columns presented degeneration. The pia was thickened on the posterior part of the cord in the upper thoracic region. The kyphosis, the flattening of the thorax and the weak thoracic breathing were believed to be due to a paretic condition of the muscles supporting the vertebral column,—a condition resulting from degeneration of nerve roots. The changes within the spinal column were also thought to be the result of the degeneration of the posterior roots, and this degeneration of the roots was attributed to chronic meningitis and adhesion of the dura to the spinal ganglia. The kyphosis and rigidity of the vertebral column were regarded as secondary in origin. The primary lesion was a localized affection of the spinal membranes. SPILLER.

187. SOME UNCERTAINTIES IN THE DIAGNOSIS OF CEREBRAL TUMOR. Ernest Septimus Reynolds. (British Medical Journal, No. 1899, page 333, Febr. 11, 1899.)

The author groups the uncertainties as follows:

A. DISEASES WHICH MAY SIMULATE BRAIN TUMOR.

Chlorosis, when there is optic neuritis, intense headache and perhaps hysterical motor and sensory symptoms. *Uremia*, with intense headache, optic neuritis, convulsions, especially if they are unilateral, and coma. *Chronic lead poisoning*, if associated with lead encephalopathy, convulsions, delirium, severe headache, optic neuritis, coma, and possibly acute mania. The difficulty is enhanced if there be paralysis of the cranial nerves, which may occur from plumbism. *Hysteria*, when there is headache, vomiting, convergent strabismus and various motor and sensory disturbances. *Reflex epilepsy* caused by irritation of the dura mater, or pressure of old scars, or other irritation in the region of the fifth nerve. *Anomalous cases* in which tumor was apparently present, but in which patients have recovered without an operation, or have recovered after the operation at which nothing was found. Under this head the author relates what he considers "two remarkable cases." The first seems to have been a plain case of infantile cerebral paralysis with localized fits extending over many years, and the second was very apparently a case of arterio-sclerosis, or at least one of senile epilepsy with no particular features, and the reviewer can scarcely agree with the writer that the nature of the irritation in

these two cases seems a mystery. *Syphilis*, with severe headache and implication of the cranial nerves, especially if there be syphilitic meningitis, local convulsions and optic neuritis. *Meningitis*, *abscess*, and *polioencephalitis* may simulate tumor.

B. SYMPTOMS DUE TO BRAIN TUMOR MISTAKEN FOR OTHER DISEASES.

Hysteria, when the tumor occurs in young girls, and there is no optic neuritis and no paralysis of the cranial nerves. *Reflex epilepsy*, if there has been an injury or tumor of a nerve. *Hydrophobia*, if there has been a comparatively recent dog bite. *Senile dementia* may be simulated by tumor in old people. When symptoms of tumor come on suddenly, they may be assigned to *circulatory changes*. If *ear disease* be present, or has existed, the symptoms may be ascribed to abscess or meningitis.

C. UNCERTAINTY IN THE DIAGNOSIS OF THE POSITION IN CEREBRAL TUMOR.

The uncertainties enumerated are too familiar to all neurologists to require repetition.

D. UNCERTAINTY IN THE NATURE OF THE TUMOR.

This is so notorious as scarcely to need mention. PATRICK.

188. UEBER MAGEN-, DARM- UND HARNBLASENCONTRACTIONEN WAHREND DES EPILEPTISCHEN ANFALLS (Concerning Contractions of the Stomach, Intestines and Bladder During the Epileptic Attack). W. Ossipow (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, Nos. 1 and 2, p. 94).

Ossipow performed a number of experiments on dogs to determine the action of the abdominal viscera during the epileptic attack. The convulsions were produced by faradic irritation of the motor area of the brain, or by the intravenous injection of the essence of ab-sinthium. The abdomen was opened and the contractions of the viscera observed. His conclusions are:

1. Contractions of the stomach, intestines and bladder occur in the epileptic attack, and usually persist after the attack is over.

2. Contractions of the stomach occur in about 50 per cent. of the attacks, and are observed chiefly in the cardia and pylorus.

3. Contractions of the small and large intestines and of the bladder are constant phenomena during the epileptic attack.

(a) Duodenal contractions begin in the clonic period of the attack, or shortly after the cessation of the spasms of the striated muscles.

(b) Contractions of the small intestine begin in about the middle of the clonic period of the epileptic attack.

(c) Contractions of the large intestine begin either in the tonic or the clonic period, more commonly at the commencement of the clonic period.

(d) Contractions of the bladder occur usually at the commencement of the tonic period.

4. The contractions of the intestines and bladder are very vigorous, and appear as long-continued spasms. This is especially true of the contractions of the large intestine and bladder.

5. Relaxation of the intestines and bladder, of longer or shorter duration, usually occurs between two vigorous contractions, and after the cessation of all epileptic contractions.

6. The phenomena relating to the stomach, intestines and bladder, observed in the epileptic attacks produced by faradic irritation, are not the result of the local irritation of the cortical centers of the stom-

ach, intestines and bladder; but are the result of the epileptic attack that develops from the irritation of the motor area of the cortex and the centers contained within it.

7. The asphyxia occurring during the epileptic attack favors the contractions of the stomach, intestines and bladder.

8. The great pressure of the diaphragm and of the abdominal wall on the stomach, intestines and bladder and their contents, during the convulsions, appears also to stand in causal relation to the contractions of the organs in question.

9. The discharge of urine and feces, occurring so often during the epileptic attack, are the result of the contractions of the intestines and bladder and of the pressure exerted by the abdominal wall.

10. The epileptic contractions of the striated muscles and the contractions of the stomach, intestines and bladder during the epileptic attack, originate in the motor area of the cortex. SPILLER.

189. SULL'ACROPARESTESIA (ACROPARESTESIA E ACRODINIA). (Concerning Acroparesthesia.) Luigo Ferris. (Il Morgagni. 4, 1899, p. 201.)

After reviewing the literature on this subject the author attempts a classification of the morbid condition under three principal forms: (a) acroparesthesia in which a disturbance of the sensibility is the only existent phenomenon (cases of Bernhardt, Laquer, certain cases of Schultze); (b) acroparesthesia with vasomotor phenomena which may be in the nature of a vaso-constriction with paroxysmal pallor of the skin (Nothnagel), or of a vaso-dilatation with reddening, heat and excessive sweating (Strümpell), or with a form of acrocyanosis, as in certain cases of Schultze and in the cases of Rosenbach after several relapses; (c) acroparesthesia with phenomena of disturbed nutrition (Rosenbach's form, certain cases of Schultze).

Ferrio then reports a personal case embodying an unusual fo.m. It was that of a previously vigorous bricklayer, 24 years of age and married. The family and previous history were unimportant. The patient was a wine-drinker, but did not use tobacco. The present illness was of a month's duration and came on while the patient was in full health. The first intimation was an intense and tormenting pruritus in the palms of the hands and in the soles of the feet without any eruption or skin alteration in those regions, but after ten days both hands and feet began to swell, especially in those parts which were the primary seat of the pruritus. The swollen areas were cold, livid and constantly bathed in perspiration; movements of the extremities were inhibited and the patient was incapacitated for all work. The pruritus and formication persisted and an eruption of small reddish macules appeared on the palms of the hands. Coincident with these first sensory disturbances on the extremities there was noted on the radial side of the carpus, in the axillary region and on the flanks a bullous eruption with clear contents, which broke down, leaving discolorations. All these phenomena disappeared after a fortnight. A few days later, however, the entire face, even to the hairy margin of the scalp, became blue and swollen, and was the seat of a pruritus, though not so intense as that in the hands and feet. The pharynx and larynx were not involved. There was a slight evening rise of temperature. At the end of a second fortnight the swelling of the face had considerably diminished, but the patient continued to suffer from a persistent feeling of formication in the hands, which were constantly cold. Examination at this time revealed a diffuse swelling of the face, most marked in the lips, in the alae nasi, in the intersuperciliary region and in the pinna of the ear. The skin over the swollen

areas was red and shining, and did not pit. Nothing remarkable in the nasal fossæ in the ear, or eye, except a slight hyperemia of the palpebral conjunctiva. The tongue was moist, not coated or swollen. The gums were slightly swollen and spongy. Nothing in the pharynx. The soft structures of the hands were the seat of a hard and elastic swelling which did not pit nor retain the imprint of the compressing finger. This was most marked over the thenar and hypothenar eminences, at the level of the pulps of the finger-tips and over the palmar surface of the phalanges, while the dorsal surface showed almost nothing. The skin of the hand was pallid, slightly cyanotic, moist and cold. Over the thenar and hypothenar eminences were noted darkish-red and livid punctate spots as large as the head of a pin, which did not disappear on pressure. No trophic disturbance in the nails. The palms of the hands as well as the palmar surface of the fingers was the seat of a constant and annoying formication, which at times assumed a burning character and was subject to variations in intensity without reference to the time of day. The forearm was never invaded. In the feet the same annoying sensations were felt as on the hands, and although the skin was pallid and cyanosed, cold and moist, it was without obvious swelling or ecchymosis. Sensation was absolutely normal throughout except the thermic; cold being perceived better than heat. Nothing was found in the internal organs except a very slight souffle with the first sound at the apex of the heart, with accentuation of the second sound at the base. No abnormality of the urine.

All these manifestations persisted more or less constantly for nearly a month, eruption succeeding eruption over various areas of the body. At this time the patient eloped.

In the matter of pathology Ferrio thinks that the changes are to be sought in the peripheral nerves, and that they probably consist of slight interstitial or perineuritic infiltrations. He regards the preservation (in most cases) of the functions of motility and sensation as evidence of the integrity of the axis cylinder. From the data collected as to the age, sex, occupation, etc., of the persons affected, he finds that the condition comes on most frequently between the ages of 25 and 50, rarely appearing after this time, and never occurring before the twentieth year. The female sex represents nine-tenths of the reported cases, the trouble attacking washerwomen and domestics more especially, but at times even the leisure classes. Among the causes, come diseases affecting the general nervous tone, and local excitants, such as alternating heat and cold. The disturbance often has a gradual onset, but at times appears suddenly. Among the curative agents recommended are electricity and massage, rest and tonics. The bowels should be carefully regulated.

COURTNEY.

190. UERER DEN GEGENWAERTIGEN STAND DER LEHRE VON DER COMMOTIO SPINALIS. (On the Present State of Opinion with regard to Spinal Commotion.) Schmaus. (Münchener med. Woch. 1899, p. 75.)

Reviewing the results of clinical and experimental researches by himself and others, the author draws the following conclusions:

1. Concussion of the spinal cord is to be distinguished from concussion of the vertebral column, since in the latter case the symptoms may depend upon direct injury of the cord or the nerve roots, and not upon true "commotio spinalis."

2. The view that there exists a true condition of spinal commotion (in the strict sense), has lost ground of late years, since there is no record of a case (in the human subject) which has been confirmed on autopsy.

3. We are not yet in a position, however, to absolutely reject all cases hitherto reported as examples of this condition.

4. The occurrence of a direct traumatic neurosis of nerve elements, through a "commotion," is experimentally confirmed, and seems calculated to explain certain facts which the reference of the lesion to direct injury of the cord leaves unexplained.

5. To tearing of the tissues through sudden gush of the cerebro-spinal fluid produced by the concussion, an important influence is to be attributed.

ALLEN.

191. RAPPORT ENTRE LA MALADIE DU SOMMEIL ET LE MYXŒDÈME.) Relations between Sleeping Sickness and Myxedema.) E. Regis and N. Gaide. (*La Presse médicale*. 1898, 81, p. 193.)

The patient, a negro in one of the French Soudanese regiments, had been transferred to the hospital on account of an irresistible tendency to sleep. This condition had persisted for three months. It commenced with severe frontal and supra-orbital pains, some vertigo, and weakness of the lower extremities. The patient's character was altered. He became irritable, melancholic, and solitary. The pupils were contracted and unequal, and reacted slowly to light and accommodation. There was salivation, some impairment of hearing, and tinnitus. The lymphatic glands were enlarged, the patient was considerably emaciated; all the reflexes were abolished, the pulse was regular, about 50 per minute, and the temperature in general subnormal. From time to time there was a tremor of the right side of the body, which occasionally became exaggerated into a convulsion. He was placed upon thyroid extract, which in the course of three days produced a very remarkable amelioration in his condition. The headache disappeared almost completely, the condition of torpidity diminished, the diuresis diminished, and the patient expressed a desire to take exercise. On the fourth day, however, he was seized with a very severe convulsion and died. This treatment was suggested by the analogies that Gaide observed between the cretins in Savoy and other cases of sleeping sickness that he had seen in the French Soudan. It appears that this disease belongs to the toxic infectious group. The enlargement of the thyroid and lymphatic glands appears to be secondary and not primary.

SAILER.

192. REMARKS ON THE DIFFERENTIAL DIAGNOSIS OF INSULAR SCLEROSIS FROM HYSTERIA. Thomas Buzzard. (*British Medical Journal*, No. 2001, p. 1077, May 6, 1899.)

The author says that the most frequent of all modes by which insular sclerosis is ushered in is by a loss of power, often of very sudden origin, being complained of in one of the limbs. This may or may not be accompanied or preceded by a feeling of "numbness" or "tingling." The loss is only partial; the patient lets things drop from the hand, perhaps, or what is still more common, in the course of a walk drags one of the legs. The limb, however, is still employed, though awkwardly. It is not uncommon for this to occur in the sequel of a strong mental emotion, and it is very likely that in the course of some days or in a few weeks the power is regained. In these circumstances, especially if the patient be a young woman, the symptoms are pretty sure to be set down to hysteria.

In not a few cases an occurrence of squinting (paresis of an external ocular muscle) ushers in the disease, and the attacks of this kind are usually transitory, like those of the extremities. This is a symptom which, according to the author's experience, is not one of hysteria.

It sometimes happens that blurring of the sight in one eye, which may go on to considerable loss of vision, is the first symptom, or it may

occur later. The ophthalmoscope may show no change. Under the idea that the symptom is due to hysteria, the patient is encouraged to expect recovery, and this will very likely happen in due course (like the recovery of power in a limb). It will, however, be liable to be followed some time afterwards by a similar affection in the other eye, or a return of the difficulty in the same one. Queerly enough, the author cannot remember having seen a case thus characterized which proved to be one of purely functional disease.

He says that in at least fifty per cent. of cases of multiple sclerosis he has found some atrophy of one or both optic disks; and, further, that the patient whose fundus exhibits no change may have considerably more impairment of visual power than one in whom there is found a distinct though partial atrophy of the disk.

He is positive in the assertion that well-pronounced and persistent ankle-clonus is a symptom of some structural change in the pyramidal tract, and that absence of knee-jerk with ankle-clonus must be taken as clear evidence of organic disease. It is not likely that the experience of all neurologists would agree with that of the author in the absence of a single well-marked example of intention tremor in a case of purely functional character. Others would probably agree with him in saying that scanning speech is not a very uncommon feature of insular sclerosis. Anesthesia of the lower extremities, if very complete, is indicative of hysteria, and contracture of the arm is of similar significance. In by far the great majority of cases of hysterical paraplegia, even those in which anesthesia is either absent or very slight, the plantar reflex is absent. As this reflex is nearly always present in cases of multiple sclerosis, the symptom is of considerable value.

PATRICK.

193. MALADIE DU SOMMEIL ET MYXŒDEME. (Sleeping Sickness and Myxedema.) C. Mongour. (*La Presse Médicale*, 1898, No. 78, p. 472.)

This exceedingly interesting case occurred in a butcher 50 years of age, a man in robust health, weighing 110 kilograms. He would be called at four in the morning, arise and attend to his business until about ten o'clock. During this period, while occupied he was apparently normal, but if he permitted himself to sit down or even stood without occupation, he would fall profoundly asleep. As soon as he had finished his breakfast he retired to his room for a long nap and repeated this after dinner. It was exceedingly difficult to arouse him at any time, and when aroused he was stupid and heavy for some time. Physical examination showed absolutely no lesion to account for this condition. The face was perhaps very slightly swollen and had a round, moon-like appearance. Partly because of the insufficiency of other treatment, partly to reduce the excessive obesity, thyroid extract was employed. The weight did not diminish, but the tendency to sleep disappeared completely. It reappeared, however, after interruption of the treatment, and disappeared anew when this was again begun.

SAILER.

THERAPY.

194. AUTOTHÉRAPIE PSYCHIQUE. (Psychical Auto-therapeutics). P. E. Lévy. (*La Presse médicale*, 8 Octobre, 1898. No. 83, p. 210).

The author suggests that there are certain periods in our existence when we are more susceptible to suggestion than in others, and it may have a large effect upon the formation of the character if we take advantage of this fact. The most favorable period, according to him, is when the subject is just about to fall asleep or is partially awakened, but still dozing. On these occasions auto-suggestion may be made with considerable effect. It is important that the suggestions should be expressed not as desires but as simple affirmations.

SAILER.

195. SEQUEL TO A CASE OF ACUTE TETANUS TREATED BY INTRACEREBRAL INJECTIONS OF ANTITOXIN. William F. Gibb. (British Medical Journal. No. 2009, page 9, July 1st, 1899.)

The author reports the death of the foregoing case about eight weeks after the last intracerebral injection, from abscess of the brain. Symptoms which are to be referred to the abscesses were present for about four weeks preceding death.

On section of the brain substance abscess cavities were revealed on each side, situated deeply in the center of each lobe. That on the left was about the size of a hen's egg and contained about 2 oz. of thick yellow pus. This cavity was in communication with the left lateral ventricle, into which pus had passed; it also communicated through the great transverse fissure with the cerebellar fossæ. The cerebellum was bathed in pus, which had also passed into the perimedullary spaces. The abscess cavity proper was surrounded by a zone of deeply congested cerebral tissue, which could be traced posteriorly backwards and downwards toward the internal capsule. On the right side the abscess cavity was about half the size of that on the left, and its walls showed less evidence of acute inflammation. It contained about 1 oz. of pus. The brain seemed otherwise normal.

Bacteriological Examination of the Pus.—Cover-glass specimens of the pus were stained with gentian violet and Loeffler's blue, and examined microscopically. Cocci were present in considerable numbers, singly and in groups. No chain cocci, diplococci, or bacilli were seen. Gelatine plates and tubes were inoculated and kept at room temperature. Agar-agar capsules and tubes were also inoculated and kept at 37° C. In every case pure cultures were obtained possessing the characteristic appearances of *staphylococcus pyogenes aureus*.

Every care was taken to secure asepsis while drilling the skull and injecting the serum; the drill and needle were always boiled before use, and as sepsis was not at any time observed clinically, it seemed difficult to believe that the source of infection was introduced with the serum; but whatever may have been the exciting cause of sepsis, damage to the brain from the repeated injections would unquestionably predispose to it. In this case repeated injections were risked on account of the excessive severity of the symptoms; probably, however, it is unsafe to venture a repetition of the process in any one case.

PATRICK.

196. PARALYSIE FACIALE GUÉRIE EN TROIS SEMAINES PAR LE SALICYLATE DE SOUDE. (Facial Paralysis cured in three weeks by sodium salicylate). Catrin. (La Presse médicale, October 8, 1898. No. 83, p. 209).

Catrin reports a case of a boy, nineteen years of age, who had a severe attack of articular rheumatism, involving nearly all the joints. Four months later he noticed that the tears from the left eye ran over the cheek, and, in the course of three days his mouth became twisted when he attempted to drink. The following day there were severe pains in the left frontal region, and a left facial palsy was discovered. This was peculiar, for the following reasons: The angle of the mouth on the left side, instead of drooping, was turned slightly upward. This could not be produced by glosso-labial spasm, because the tongue was easily protruded, and the cheek was in a state of flaccid paralysis. The tongue deviated toward the sound side. The soft palate deviated to the left, and when the patient attempted to swallow, only the left pillar of the fauces contracted, the right remaining relaxed. In addition, there was complete anesthesia and analgesia of the left half of the face, involving the conjunctiva and the mucous membranes of the cheek, excepting the forehead. Hearing was less perfect on the left side, and taste was lost

in the left half of the tongue. There were fibrillary contractions in the muscles. In view of the previous history a diagnosis of rheumatoid paralysis was made, and the patient placed upon salicylate of sodium, commencing with a dose of 2 grms. *per diem*, and rapidly increasing to 4 grms. *per diem*. In twelve days power was much improved and sensation completely restored. Nine days later the patient was perfectly well. Catrin regards his case as particularly interesting, in view of the anesthesia. This he explains by supposing that the facial nerve contains a variable number of sensory fibers. He takes occasion to criticise the old text-book description of facial paralysis and to call attention to the fact that it does not apply to all cases.

SAILER.

197. UEBER DIE SEDATIVE WIRKUNG DES METHYLENBLAU BEI VERSCHIEDENEN FORMEN VON PSYCHOSEN. (On the Sedative Action of Methylene Blue on Various Forms of Psychoses.) Pietro Bodoni, (Klinische therapeut. Woch. May, 1899, No. 21, p. 666).

Bodoni's original object was to study by means of methylene blue the renal functionality and permeability in various mental diseases, but as he was impressed at the very outset by the sedative action of the drug, he was impelled to continue his observations in this direction in cases with marked maniacal excitation. Fourteen cases were studied, the method of administration being intramuscular (in the gluteal region), the dose for the first injection 0.08 gramme, and for the second 0.10 gramme. The results in all were immediate and striking, and were manifested by a cessation of previous excitation and the production of a sleep of several hours duration. The form of the psychosis did not seem to influence the action of the drug in the least. The pulse, temperature, respiration and urine were watched throughout the course of administration, and were not found to be disturbed. Special emphasis is laid by the author on the necessity of using methylene blue which is chemically pure, as he believes the undesirable sequelæ reported from its use in the hands of other observers to have been due to impurities. In its sedative action he places it on a plane with sulfonal, Trional, Tetronal, Chloralose, Duboisin, Hyoscyamin and fonal, trional, tetronal, chloralose, duboisin, hyoscyamin and bromoform.

COURTNEY.

198. DEUX CAS DE PARALYSIE RADICULAIRE OBSTETRICALE DU PLEXUS BRACHIAL; EXAMEN ET TRAITEMENT ELECTRIQUE (Two Cases of Obstetrical Paralysis of the Roots of the Brachial Plexus; Electrical Examination and Treatment). Felix Allard (La Presse médicale, September, 1898, No. 79, p. 177).

Allard, after calling attention to the infrequency of this lesion, reports two cases. The first, a boy eleven years of age, had had at birth an exceedingly large head. This had required some force, but had not necessitated the application of the forceps. Immediately after birth, partial immobility of the right arm had been noticed. When examined at the age of three months, the right arm was immobile, it could not be flexed, but could readily be extended. The movements of the hand and fingers were normal. The humerus was in a condition of forced internal rotation, and the forearm in a position of pronation. There were neither disturbances of sensibility nor contractures. Electric examination showed normal electro-irritability of the fifth and sixth roots on the left side; diminished electro-irritability of the same roots on the right side. The muscles supplied with them required stronger currents than normal to produce contraction, but there were no reactions of degeneration. Treatment by the galvanic current, which alone produced contraction, resulted in complete cure. The second case, also a male child, had been extracted with forceps. Dur-

ing labor, the head had been forcibly flexed toward the left shoulder to disengage the right, producing considerable tension upon the fifth and sixth roots on the right side. The results of the electrical examination were identical with those in the previous case; quantitative diminution but not qualitative alteration. Both cases recovered completely after prolonged electrical treatment. Allard believes that the prognosis is favorable if true reactions of degeneration are not present. SAILER.

199. CURE OF MORPHINE, CHLORAL AND COCAINE HABITS BY SODIUM BROMIDE. Neil Macleod. (*British Medical Journal*, No. 1998, Ap. 15, 1899 p. 896).

In the *British Medical Journal* of July 10th, 1897, the author reported two cases of morphine habit cured by enormous doses of sodium bromide (1) and now reports one case of chloral habit, one of morphine habit, and two in which cocaine was used with morphine.

The chloral habitué (a Chinaman, aged 32 years), was given three drachms of bromide in four days, after which he slept for eight days, taking about two pints of milk daily. When able to move about he still exhibited motor, mental and emotional feebleness with delusions of fear and persecution. He went through a window and sustained a fracture of both bones of the left leg. Some five weeks after the administration of the bromide, he had quite recovered from its effects and remained cured of the chloral habit.

The morphine case was a lady, aged 48, of a decidedly neurotic family, and herself a marked neuropath. She took daily an average of 12 grains of morphine hypodermically, and this dose had been taken the day on which treatment was begun. She was given six drachms of bromide between four and six p. m. The next day she was given 6 grains of morphine in two doses, and received during the day 9 drachms of bromide. On the third day 2 grains of morphine were injected and one ounce of bromide was given between 9.00 a. m. and 10.30 p. m. After this no medication was used save a laxative pill, and she slept for five days except when roused to take milk and for the evacuation of bladder and bowels. Marked physical and mental hebetude with numerous delusions continued for two weeks, followed by complete recovery.

One of the morphine-cocaine cases was seen in consultation, and is not reported in detail. The plan of treatment was the same, the patient being given a large quantity of bromide for three days, which was followed by sleep for four or five days, after which the craving for drugs ceased and the patient was considered cured.

The other case of mixed addiction was a physician who had already reduced his daily dose from 30 grains of morphine, and 15 to 20 grains of cocaine, to 5 grains of each. On the evening of the first day of treatment, having $6\frac{1}{2}$ grains of morphine, he received 1 ounce of bromide in doses of 2 drachms. The second day he was given $2\frac{1}{2}$ grains of morphine and 10 grains of bromide, and the third day $\frac{3}{4}$ grains of morphine and drachm doses of bromide at intervals through the night. On the fourth day he was given an injection of 1 grain of morphine and no bromide. He received no drugs after this. On the fifth day he developed pneumonia of which he died two days later.

The author concludes: (1) The withdrawal within three days of even large doses of the drug causing the habit; (2) the certainty that this will cause no suffering; (3) the patient cannot deceive those dealing with him in the matter of secret administration, nor can he enlist after the third day the aid of attendants; (4) any physician with the aid of vigilant nurses can deal with the case in any hospital or private house, no special institution is needed; (5) there is no risk of substituting another drug habit, and the craving will be lost whether the patient desires it or not.

PATRICK.

Book Reviews.

UEBER DAS PATHOLOGISCHE BEI GOETHE. By P. J. Moebius, published by Johann Ambrosius Barth, Leipzig, 1898.

The little volume before us consists of an interesting application of the principles of psychiatry to the study of Goethe and his writings. Not that Moebius has followed in the foot-steps of Nordau and decried the great works of the man who is the subject of his study, but merely that he has made a scientific and scholarly analysis of the facts presented by Goethe's works and his life, and thereby again directed attention to the interesting parallelism so often seen between genius and insanity, as well as to the narrowness of the line of demarkation that separates these two conditions. The book is divided into two portions—first, Goethe's works, and second, Goethe's person.

Moebius calls attention to the differences in the points of view which naturally obtain when insanity is regarded by scientific men and by poets, and further shows that in the portrayal of the passions and emotions of men, the poet is naturally drawn toward the pathological. The more a poet is a true mirror of reality, the greater will be the rôle which the pathological will play in his writings. In proof of this he points to the numerous pathological characters in the writings of such poets as Shakespeare and Goethe.

As regards the observation of the ordinary insane, Goethe appears to have had but few opportunities, nor does he seem to have improved those which offered themselves. He had an intense dislike of the madhouse, and on one occasion when an inducement was held out to him to visit one of these institutions, he said that he did not in the least desire to see the fools who were locked up, inasmuch as he had more than enough of those who go about at liberty, and further declared himself to be ready to follow the "Duke to hell if need be, but not to madhouses." He interested himself considerably in medical matters, and he frequently had the opportunity of conversations with physicians and medical professors in the universities. For instance, he was well acquainted with Heinroth who taught clinical psychiatry in the early part of this century, but it does not appear that Goethe especially interested himself in Heinroth's subject. The material of the pictures which Goethe gives in his writings of diseased mental states must be regarded as having been drawn from actual observations, from occasional conversations and perhaps occasional lectures. He had various opportunities for observing people presenting gross mental disturbances. A young man afflicted with dementia lived as ward in his father's family. Rechtscandidate Clauer appears to have been the original of the young lunatic in "Werther's Leiden." Again, Lenz, although he was not pronouncedly insane, presented during his illness peculiarities which could not help but impress Goethe. Of significance was also Goethe's relations with the physician Zimmermann. Zimmermann was possessed of a morbid nature and later on became actively insane.

Goethe used the various terms of insanity very much as did other writers of his time. Thus he used the word *hypocondria* as applied to irritable, gloomy, nervous or melancholic people or persons suffering from depressive delusions. Goethe used the word *melancholia* at times to designate depressive emotional conditions in persons who are otherwise well, and at times to designate morbid and sad emotional states. The word "*Wahnsinn*" he used mostly as lay people use it to-

day, namely as signifying insanity in general. The word "Narheit" (folly) he used as silliness or dementia. If the madman is greatly excited, especially angrily excited, Goethe speaks of "Tollheit" (rage) or "Raserei" (fury). The word "Verücktheit" he used very seldom, and apparently in the sense of insanity in general. Goethe also makes use of the expression "Seelenleiden" (ills of the soul).

Moebius takes up Goethe's writings in some detail; e. g., "Werther's Leiden," "Lila," "Faust," "Iphigenie," "Tasso," "Wilhelm Meister," "Benvenuto Cellini," "Wahrheit und Dichtung," "Wahlverwandschaften," "Wanderjahre und kleinere Erzählungen über das Wunderbare," und "Allegemeines und Einzelnes;" in each instance pointing out the pathological features presented by various characters. He says, reviewing the characters of Goethe, we find, disregarding the historical representations, only in a few a picture of morbid psychic conditions which is true to nature. Lila, Orest, the harper Mignon are purely creations of Goethe's fantasy. Representations according to nature are only found in the young lunatic in Werther, in a certain sense in Werther himself, in the crazy count and (in spite of Goethe) in Tasso. However, that which is of importance for us consists in the fact that Goethe, without any theoretical education, was impressed with the significance of pathological conditions—so much so that he, more frequently than any other author, pictures them. Again, he especially and accurately describes transitional states. He evidently had abundant opportunities to observe these among the numerous pathological men and women whom it was his fortune to know.

Goethe evidently regarded the passions as the cause of psychic disturbances. By the excitation of the passions a level may be reached where transition into insanity takes place. In this manner he speaks in various places of his own passions. He also makes Werther say, "My passions were never far from insanity." Concerning his ideas of the treatment of the insane we have the evidence of the country parson to whom the harper is brought. The parson says, "Outside of the physical condition that often presents unsurmountable difficulties and for which I always call to my assistance a physician, I find the means to cure insanity very simple. They are the same by which we prevent well persons from becoming insane. We should excite the spontaneity of the patients, we should accustom them to order, we should give them to understand that they share their existence and their fate with many others in common, and that the most remarkable talent, the greatest good luck and the greatest misfortune, are only slight departures from the average; then no insanity will creep in and if it is present, it will little by little disappear." It is noteworthy that the parson's conception was a humane one, which took no account of the barbarous measures commonly practised in Goethe's time. Goethe's conception regarding the nature of insanity is further revealed in a remark made to Hegel. At tea they were discussing dialectics, and Goethe thought that the latter were often misused to make false things true and true things false. Hegel had the assurance to reply that this misuse was only perpetrated by people who were ill. Instead of answering with Nathan "Thou art the man," Goethe said with good-natured irony, "Such dialectic patients can find their cure in the study of nature." On another occasion Eckermann said, "One so frequently finds associated with brilliant talents, especially in poets, a feeble constitution." "The extraordinary things which such persons accomplish," answered Goethe, "implies a very delicate organization, one which can perceive rare sensations and hear the voices of the heavenly. Such an organization is, however, in con-

flit with the world, with the elements. It is easily disturbed and injured, and he who does not like Voltaire unite great sensitiveness to excessive robustness, is easily subjected to prolonged ill health. Schiller was continuously ill. When I first saw him I thought that he would not live four weeks, but he also had a certain toughness. He lived many years and could with a healthier manner of living have lived longer." Goethe added, "There was a time when in Germany one thought of a genius as little, weak and even humpbacked, but I prefer a genius with an appropriate body."

The most interesting part of this little volume is that which deals with Goethe himself. Moebius speaks of Goethe's family. Goethe himself declared that he had his father's constitution. In his features he resembled his mother, especially as regards the eyes and the mouth. The father possessed stability and solidity, qualities which expressed themselves in an earnest desire to learn and teach, in a strong love of order, in conscientiousness, in a disregard of himself, in an absence of wants and in self-repression. The same virtues are discernible in the son. In the father they were dimmed by a certain narrowness which made him appear pedantic, self-willed and inconsiderate toward his family. In contrast with his son he was subdued and unimaginative. Avaricious, suspicious and morose, he made life difficult for himself and those about him. In his old age he broke down rapidly, became demented and passed his last years in a pitiable condition. There can be no doubt that he was in a marked degree pathological. The qualities of the mother appear to have been those active in making a poet of the son. She was sensitive, gifted with a warm imagination, cheerful and possessed with an unconquerable love of life. Pathological factors appear to have been present in a slight degree if at all. Goethe's sister Cornelia was so much like her brother that at one time they could have been regarded as twins. She was, however, lacking in both physical and mental attractiveness. The very features which made her brother's face so attractive, *e. g.* the strong nose and high forehead, made her face repellant. Her attitude also was bad, and she appeared to have suffered from an eruption which especially seemed to make its appearance whenever she went to a ball. Goethe says that she was also without sensuousness, that she had a disgust for the marital relation, and therefore, lived unhappily with her husband. Goethe praises her virtue and her sound and acute intellect, but added that she had a firm, hard and almost loveless manner. "My sister," he says, "was a remarkable and indescribable character, and a most strange mixture of strength and weakness, of self-will and pliability. One could say of her that she was without faith, love or hope." She was reserved, became hypochondriacal, at times violent and passionate, at other times dull and indifferent. Her engagement and marriage with Schlosser appeared to have temporarily raised and enlivened her, but her happiness was of short duration for she was not capable of love. The three and a half years of her married life appear to have been a period of suffering for both parties. She finally died in a second confinement. The unhappy Cornelia appears to have been of a thoroughly pathological constitution. Concerning the other sisters and brother of Goethe, all of whom died early in life we know but little. One brother Jacob lived to be six years of age and then died of a contagious disease. One sister lived to be two and a quarter years old, and another lived only seven months.

Goethe, asphyctic at birth, developed for a time normally. As a child he appears to have been precocious, lively and cheerful. He seemed later to have suffered from various infectious diseases, among

them, smallpox. Little else is known up to his obscure illness in Leipzig. Here he evidently suffered pain in the chest and was hypochondriac. His temperament, as he himself relates, wavered at this time between the extremes of unrestrained merriment and melancholic depression. In writing of this time he gives a vivid description of the various sensations that he experienced, so much so that Moebius speaks of this description as the anamnesis of a hypochondriac. He also suffered from an exhausting hemorrhage from the mouth, and for several days hovered between life and death. Moebius, endeavoring to explain this hemorrhage after disposing of hemorrhage of the lungs and ulcer of the stomach, suggests as a possibility a bleeding of nervous origin (?). Goethe recovered rapidly, and subsequently returned to Frankfort where, to his father he appeared like a patient whose illness was "more of the soul than of the body." A long period of invalidism followed; he apparently presented a neurasthenic hypochondriac syndrome, a "Nervosität," as Moebius puts it.

He was further subject to periods of exaltation, and these were so marked as to suggest to Moebius maniacal excitement or hypomania. That his emotional excitement and irritability must have been very great is drawn from his own writings, for Goethe, the youth, says of himself, "Could you see this miserable wretch, how he rages, but does not know against whom he should rage, you would pity him. How can a madman become reasonable? That is I. Chains on these hands! Then I would know into what I should bite." Contemporary accounts tell how the young Goethe expended his anger by breaking pictures on the corner of his table, by shooting his books, and so on. It is well known that Goethe also suffered from violent explosions of excitement in later life. Other distinct pathological elements are also discernible in Goethe's youth. He at times felt a disgust of life and entertained ideas of self-destruction. To Eckermann he said in his old age that he never read "Werther's Leiden" but once. "It is made up of fire rockets. It makes me gloomy, and I am fearful of again living through the pathological condition which gave rise to the book." On another occasion he said, "When the *tedium vitae* seizes a man he is to be pitied *not* to be blamed. That all the symptoms of this fearful disease, at once natural and unnatural, have raged in my innermost self, this story of Werther will leave no one to doubt. I know very well what effort it cost me at that time to escape the waves of death just as it did to save myself from many a later shipwreck." Concerning this state, Moebius writes, "The *tedium vitae* of youth is evidently a phenomenon of all times and peoples. The young Buddha saw that nothing in life was enduring, and he renounced the world! The young Schopenhauer wrote his fourth book concerning will and idea! How many young men may in the two thousand odd years that lie between Buddha and Schopenhauer have passed through similar suffering! One could say that it is peculiar to highly gifted human beings to experience an especial pain when they discover the chasm between the ideal and the barrenness of the real world, and most acutely at the time that this discovery is first made. The spiritual fever from which Goethe suffered did not die with the writing of Werther. Slowly it disappeared in the next few years only to again flare up at subsequent periods—love affairs often playing the rôle of irritating causes. However, a gradual subsidence took place and Goethe himself realized the grateful restfulness that ensued. In Goethe's manhood pathological elements were present only to a minimal degree. His irritability it is true, still persisted and occasionally led to periods of excitement. That he was constantly emotional is evidenced by the fact of the remarkable ease with which he wept.

As already stated, following the first period of excitement, Goethe passed into a period of increasing clearness of mental vision and quiet. This period was interrupted at various times by a serious recurrent excitement which, as Moebius points out, corresponded with his periods of poetic productiveness. Goethe himself felt very clearly that the desire to write poetry came upon him as a fever, that at certain times he had to compose, and that after the disappearance of the excitement the well of song was dry. Goethe describes how on awakening at night verses or songs would teem through his mind, and how he would hastily rise and without waiting to arrange his paper would dash down his thoughts. His numerous love affairs also bore a distinct relation to these periods of excitement, and his passion frequently expended itself on women who were neither attractive mentally nor physically. He loved not because he was in the proximity of an attractive woman, but because the period of excitement was upon him. Moebius says that the recurrences of periods of excitement can hardly be termed periodic, yet it is noteworthy that the duration of these periods was as a rule the same, namely upwards of two years. That Goethe understood himself very well is evidenced by a remark made to Eckermann, "Such men and their like possess the constitution of the genius; their state is peculiar to themselves. They experience a *repeated puberty* while other persons are young but once."

The domain of the pathological is also touched upon by the doctrine of the two souls; the expression is Goethe's own. He himself is at one and the same time Faust and Mephistopheles, emotional and excited and the critic. Herman Grimm in speaking of these qualities of Goethe says: "As far as we know Goethe never experienced anything which made him lost completely to himself. Even when he was most passionately roused, he always retained the strength to quietly criticize himself. Experience and subsequent reflexion must always be separated in him. When Goethe was separated from Frau von Stein, he expressed himself more passionately than at her side. Only when he fully reflected did his passion find expression. We have seen how his relation to Lotta only became comprehensible when we relegate his passion to the times when he is not with her." "He who is able," says Moebius, "at such times to observe himself, is well fitted to be a painter of souls, but he is not normal."

Moebius also discusses the various bodily ills of Goethe. Goethe was ill quite a great deal. In addition to the nervousness and hypochondriasis of his youth, he suffered at various times from influenza, renal colic, erysipelas, cough, angina, pleurisy, hemorrhages and persistent disturbances of digestion. As a personal peculiarity of Goethe it is mentioned that he preferred the close atmosphere of unventilated rooms, just as Schiller found a special inspiration in the odor of rotten apples. Goethe was also said to have been exceedingly irritable regarding order in his room. For example, he could not stand it when a book was lying crooked upon his table, or if someone other than himself trimmed his light.

Moebius dwells upon the wonderful productiveness of Goethe in spite of these drawbacks. He says, "At no time do we realize so clearly the fulness of this man's life as when we view the unremitting work and the insatiable desire to learn still manifested in his clear and cheerful old age. He was a man and had to grow old, had to lose his power of production, and had to betray both in thought and word his age, as well as in his skin and hair; but in spite of all there burned in this old man a flame of which every youth could have envied him. Faust is indisputably a marvelous production. The early Faust had his origin in the period of fermentation. Passionate excitement, pessi-

mistic tendencies, tempestuous desire to know, ruled him in the first scenes; highest poesy in the Gretchen scenes. To the early Faust the later Faust owes his power over the soul; through him the work becomes a guide and ideal to youth. Even if the parts of Faust written by the mature and aged Goethe do not attain the overmastering force of the Faust of Goethe's youth, they still belong to the most beautiful productions of the human mind. It is just these productions of the old age of Goethe that from their complete form, their wisdom and piety are in great part invaluable. When has an eighty-year-old man ever written the like."

As is well known, Goethe died a few hours after an attack of angina pectoris. He does not appear to have realized that his death was impending. The end appears to have come without suffering.

Regarding his children the following suggestive facts obtain: In December, 1789, a son, August, was born; in October, 1791, a still-born boy; in November, 1793, a daughter, who died in the following December; in November, 1795, another son who died in a little over three weeks; in December, 1802, a still-born daughter. It must be added here that Goethe's wife became some time after marriage alcoholic and later on epileptic. Moebius, however, does not attribute the pathological elements noted in Goethe's children entirely to the mother, but attributes them also to the genius of the father. August, the sole surviving child of Goethe, appears to have been talented, with features resembling his father, though his lips were coarse and were apparently derived from the mother. He began to show a tendency to alcoholism in early life and also early manifested a strong tendency for the opposite sex, and, it is said, in the company of Ernest Schiller was guilty of various excesses. He outwardly decried any interest in poetry, for as he declared he "did not desire to maintain the firm and business of his father." Secretly, however, he appears to have written poetry at various times, but his productions were not of an enduring nature. He manifested considerable interest in scientific studies and also in the collection of natural objects. He died at forty years of age at Rome, whither he had apparently gone to recover his health. As it is nowhere stated that he was suffering from any disease, it is quite probable, Moebius thinks, that he was suffering from a mental affection. Indeed his death occurred under such circumstances as to suggest the thought that he had committed suicide. Information, however, regarding the entire subject is unsatisfactory.

August's wife, Ottilie von Pogwisch, appears to have been a pronounced degenerate. She was passionate and fantastic, and was known among her acquaintances as the "Crazy Angel" and the "Woman from the Other Star." The oldest son, Walter Wolfgang, born 1818, was a quiet, submissive fellow, devoted himself especially to music, and suffered much mortification because no attention was paid to his compositions. He was devotedly attached to his mother, and sacrificed much in her behalf. Later in life he lived retired, gloomy and repressed. He is said to have been small, delicate and somewhat crippled, mentally simple and modest. He early developed consumption, but only died in 1885. Another son, Wolfgang Max, born in 1820, was in his youth serious, diffident, passionate and imaginative. He betrayed excellent talents and wrote as a young man a drama called "Erlinda." He later devoted himself to philosophical and historical studies and worked during his entire life without ever completing anything. He was for a time in the diplomatic services, and subsequently lived alone, moody and ill. He suffered greatly from neuralgia, and appears almost never to have been well. He died from an asthmatic affection in 1883.

Moebius closes this interesting little volume with the following paragraph: "It is said that families, like individuals, have a definite

length of life. The stock of Goethe has withered. His family bore in him an exquisite flower, and in so doing exhausted its strength; after him there followed only weak and feeble off-shoots. Genius appears on the earth, not to increase the number of mankind—its works are its immortal children."

Moebius' interpretation of Goethe impresses the medical reader as not only scientific, but in all probability as correct. There is little to criticise in the picture he has drawn.

F. X. DERCUM.

ATLAS OF THE EXTERNAL DISEASES OF THE EYE. By Prof. O. Haab, Zurich. Edited by G. E. de Schweinitz, A.M., M.D. Pp. 228, with 76 colored plates and 6 engravings. Philadelphia; W. B. Saunders. 1899.

The artist who made the colored drawings for this book in the main, did his work well and the plates compare favorably with the colored reproductions of photographs in Ramsay's more pretentious and much more expensive atlas. The text, however, is less satisfactory. The descriptions of the external diseases of the eye scarcely equal those found in the ordinary text book and they are often very brief, while many pages of the book, unnecessarily it would seem, are taken up with a description of the functional examination of the eye that is too cursory to be of much value. There is, too, a suggestion of provincialism in the author's tendency to confine himself to the nomenclature and methods of treatment in vogue at the Zurich clinic, although this is relieved by the more catholic notes of the American editor. The translation is fairly well done, but it would have been only charitable on the part of the translator to have corrected such minor blunders as "the capsular *endothelium* of the lens" (p. 114). The excellence of the plates and the low price of the volume will commend it to those who are interested in the external diseases of the eye, but lack the opportunity of attending large clinics. HOLDEN.

THE
Journal
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Original Articles.

FACIAL PARALYSIS, CONGENITAL, UNILATERAL AND
OF UNIQUE DISTRIBUTION.*

BY F. W. LANGDON, M. D., of Cincinnati.

A. F. L., thirty-three, Kentuckian, single, was born and grew to maturity on a farm. For the past five years he has been a professional nurse employed most of the time as an attendant in hospitals for the insane. In January, 1899, he presented himself to me for advice regarding some nervous weakness following la grippe, when I noted that he had a facial palsy of unusual type. The movements of the lower facial group were almost intact, whereas the left upper face presented a marked lagophthalmos and total absence of occipito-frontalis action.

On inquiry as to the duration of the paralysis he informed me that it had existed from birth, and that he was told by his mother that he had been "born so."

The Family History does not seem to throw much light on his defect. His mother was over sixty at her death which was due to cancer. His father is living at sixty-five and generally healthy with the exception of occasional "neuralgic" pains in his head. Of his fourteen brothers and sisters, one brother died at twenty after having been a paraplegic for a year; one sister died at fifteen of dropsy; three other children of the family died in infancy from unknown causes. The nine living brothers and sisters are said to be healthy, excepting that one has had inflammatory rheumatism. With the exception of the paraplegia above noted, no nervous disease or deformity is known amongst the other children.

Personal History. The physician who officiated at his birth

*Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

is not living. At my request he has recently written to his father and grandmother, both of whom were present at his birth and who are positive in regard to three things, viz.:

1. That he has never closed his left eye since birth.
2. That no instruments were used at his birth nor was the labor an unusually difficult one, he being the eighth of fifteen children.
3. He has never had a fit or convulsion or unconscious attack.

There is no history or evidence of ear disease.

He states that as he grew up on the farm the annoyance due to constant irritation of the left eye by wind, dust, etc., was so great as to compel him to seek a less exposed means of livelihood. The presence of considerable conjunctival hyperemia and a corneal cicatricial area due to an old keratitis are evidences of the effects of this irritation.

At the age of fourteen he had a moderately severe attack of rheumatism in both knees, which did not confine him to his bed. In December, 1898, a few weeks before consulting me, he had an attack described as la grippe which disease was more or less prevalent at that time.

There is no history or evidence of venereal disease.

Habits excellent as regards alcohol and tobacco.

Personal examination, January 31, 1899: Physically the patient is a large, well-proportioned man, five feet eleven inches in height, weighing one hundred and ninety-five pounds. Complexion medium; hair and mustache dark brown. Irides dark gray mottled with dark brown, plentifully at the free borders. Teeth sound but of a "gouty" type—i. e., ground off squarely and flat at their cutting edges. Temperature 97.5; pulse 120 and of irregular rythm and low tension at this examination, owing probably to his temporary indisposition. Subsequent examination when in his usual health shows a temperature of 98.6 and pulse rate of 76, fairly regular and of good tension.

A systolic bruit is audible at the cardiac apex, but does not appear to cause any somatic symptoms.

Lungs present nothing abnormal.

Digestion is usually good and bowels act normally. At the time of this examination however he had a slight diarrhea which yielded in a day or two to dietetic restrictions.

The genito-urinary system presents no defect. Urine looks normal, S. G. 1028; albumin and sugar are absent.

Mentally he is well-balanced, logical and quick of comprehension. He has a fair education.

His speech functions are normal as regards reception,

elaboration and emission of language. Articulation is not defective.

Cranial Nerves:

I. Olfaction is not defective on either side to the usual tests.

II. Dr. Robert Sattler who has kindly examined the eyes for me, reports as follows: "I can find no lesion of the fundus. The papillæ, aside from a delicate capillary injection, giving the disks a more markedly reddish hue, are normal.

"Vision is reduced in the left eye by corneal scars and the eye is enormously hypermetropic (H6). This is also probably congenital. With corrective lenses the vision in this eye is

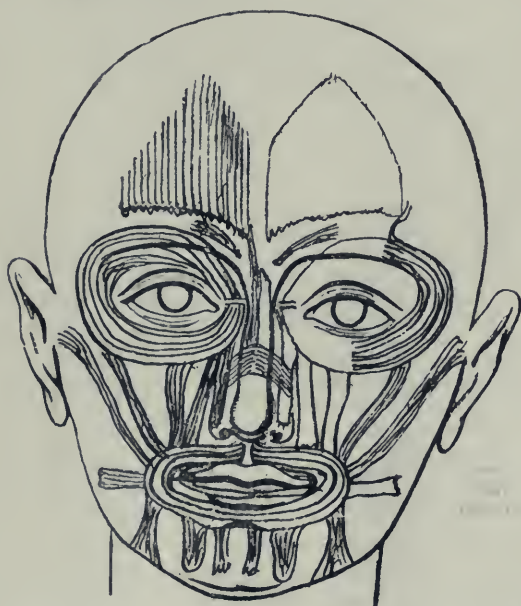


Fig. I.—Muscles in outline are absent; those shaded respond to volition, to faradism and to galvanism. R. D. is absent.

surprisingly good, viz.: 0.4. There is no defect of visual fields or color sense to ordinary examination. The right eye is practically normal."

III., IV., VI. The pupils are equal and respond well to light, accommodation and convergence. Ocular movements present and free in all directions; but in outward excursions of the globes, neither cornea quite reaches the external canthus. The left external rectus seems rather weaker than the right.

Nystagmus and diplopia are absent.

V. Taste prompt and correct in each half of tongue tested separately outside of mouth, with salt, sweet and bitter substances.

Facial sensation is excellent everywhere on both sides.

Muscles of mastication act well and equally.

VII. The following muscles of the left face fail absolutely

TABLE OF ELECTRICAL RE-ACTIONS.

O=No response, —=response diminished, N=normal. See Fig. 1.

		RIGHT.			LEFT.		
		Far.	Galv.	Qnt. Qlt.	Far.	Galv.	Qnt. Qlt.
Orbicular portion	Occipito-frontalis.....	N	N	N	O	O	O
	Orbicularis palpebrarum { inner half	N	N	N	O	O	O
	“ “ { outer half	N	N	N	—	—	KC>
Palpebral portion	“ “	N	N	N	—	—	KC>
	Tensor tarsi.....	N	N	N	—	—	KC>
	Corrugator supercilii.....	N	N	N	—	—	KC>
	Pyramidalis nasi.....	N	N	N	O	O	O
	Compressor naris	N	N	N	N	N	N
	Dilatores alæ nasi.....	N	N	N	O	O	O
	Levator labii superioris et alæ quæ nasi.....	N	N	N	O	O	O
	Levator labii sup. proprius....	N	N	N	O	O	O
	Zygomaticus major.....	N	N	N	—	—	KC>
	Zygomaticus minor.....	N	N	N	—	—	KC>
	Risoreus.....	N	N	N	O	O	O
	Orbicularis.....	N	N	N	N	N	N
	Depressor anguli oris.....	N	N	N	N	N	N
	Depressor labii inferioris.....	N	N	N	N	N	N
	Levator menti.....	N	N	N	N	N	N

to respond to voluntary effort, faradism or to galvanism: (See diagram figure 1, and table of electrical reactions); The occipito-frontalis; the inner half of the orbicularis palpebrarum, the pyramidalis, the levator labii superioris et alæquæ nasi, the levator labii superioris proprius and the risoreus. On the same side the zygomatici respond to volition, to faradism and to galvanism. A. C. <K. C. quickly, but more feebly than the corresponding muscles on the right; *i. e.*, the electrical irritability is diminished quantitatively on the left. The outer half of the left orbicularis palpebrarum also responds to volition, and to both electrical currents quite as promptly as the corresponding muscle of the right side. but also requires a slightly stronger current and its action ceases about midway of the or-

bital rim as shown in the diagram (Fig. 1). A curious diverticulum of a few fibers from its upper margin turns vertically upward as shown in the figure. The winking movements are quick, but do not close the eye, owing chiefly to inability to raise the lower lid. The tarsal muscles respond quickly to faradic and galvanic currents. The left corrugator supercilii, the compressor naris, the orbicularis oris, the lower lip depressors and the chin elevators act normally.



Fig. II.—Face at rest.

Owing to the above defects of the left facial muscles, the patient cannot wrinkle the forehead transversely on that side; the eye cannot be closed and the left nostril does not dilate in forced inspiration or on effort. The mouth is slightly drawn to the sound side, but his mustache conceals this defect quite well. He can elevate the left angle of the mouth almost as well as the right by the fairly competent zygomatici, but he cannot

"spread" the mouth laterally owing to absence of the risoreus. In other words he can smile but not "grin."

The accompanying photographs, for which I am indebted to Dr. W. E. Schenck, show some of the above described conditions fairly well. He can frown, whistle, pout, puff out his cheeks, and roll his tongue into a gutter, but this gutter seems to be unequal on the two sides, "spreading open" towards the left. The platysma responds to volition normally and equally on the two sides. All movements of the lower lip



Fig. III.—Attempt to close both eyes.

and chin are balanced and perfect. On the right face there is no defect of action in the seventh nerve group. Contracture is absent on either side of the face. A flattening in the left infraorbital and supramaxillary region is apparently due to the absence of muscles elevating the upper lip.

By measurement, the left eyelids when approximated without effort, are separated by a space of seven-sixteenths of an

inch at the widest part. Upon forcible effort this space can be reduced to four-sixteenths.

Experimentally, a course of electrical treatment (faradic, galvanic and sinusoidal, alternately) was given for a month, the ocular orbicularis and tarsal muscles being caused to contract intermittently for about five minutes daily or every other day. At the end of this period the approximation of the lids was reduced to nine-thirty-seconds of an inch ordinarily, and four-thirty-seconds on forcible effort. Coincident with this im-



Fig. IV.—Attempt to elevate eyebrows.

provement a lessening of the conjunctival injection and a subjective sense of increased comfort in the eye were noted.

VIII. Hearing is good and equal in both ears, a watch tick being heard at three feet or more.

IX. The palatal arch is higher *on the left* and the uvula slightly drawn *to the left*. The palatal reflex is active on both sides and the palate moves normally in phonation.

X. No lesion is evident in the pneumogastric distribution.

XI. The same is true of the spinal accessory.

XII. The tongue protrudes slightly to the right (the well side), but is freely movable in all directions. It presents a slight lateral longitudinal groove on the left, perhaps indicating slight atrophy; some slight fibrillary twitching is evident, more marked on the left side.

Trunk and extremities: Motion: No muscular weakness or atrophy. Gait and station normal. Grasp by dynamometer



Fig. V.—Frowning. Both corrugators act; the left less strongly than the right. The patient has also brought into action the sound occipito-frontalis, probably through a misunderstanding of what was wanted.

test, right 129, left 129 (normal about 80). Patient is right handed.

Sensation: No subjective sensory symptoms excepting more or less discomfort in left eye. No defects of tact, pain, temperature, posture or localization sense.

Reflexes: Organic: Normal.

Tendonous: Elbow-jerks, wrist-jerks and knee-jerks present, moderate and equal on the two sides.

Vaso-motor symptoms, aside from those presented by the irritable left conjunctiva are absent.

Trophic symptoms, excepting in the left face and tongue are not present.

The recent exhaustive review of the literature of congenital facial paralysis by Thomas¹ renders superfluous any reference to this aspect of the subject on my part. It may be noted, however, that of the five cases in the literature classed by Thomas as true congenital unilateral facial paralysis, only two (those of Bernhardt) are comparable with the present case. We may also quote that Bernhardt himself concludes that "although the occurrence of an isolated, unilateral, congenital facial paralysis, or, perhaps better, an incomplete development of the nerves and muscles in the distribution of the facial nerve on one side, cannot be denied, still, its occurrence has as yet not been definitely demonstrated."²

Hence, if the history of our present case be taken in lieu of actual medical observation at birth, it would seem that it is fairly entitled to rank as unique in etiology as well as in distribution.

DISCUSSION.

Dr. W. M. Leszynsky said he had recently had under his observation a patient in whom there had been facial paralysis involving all branches, and recovery had occurred in all the muscles, except the orbicularis palpebrarum, which is usually the first muscle to recover its motility. He thought that Dr. Langdon's case, if not of congenital origin, might be looked upon as one of those that had made a partial recovery. Several years ago Dr. Leszynsky reported two cases in which peripheral facial paralysis had existed for two years, and yet the faradic irritability was preserved in the paralyzed muscles.

Dr. H. M. Thomas believed that two cases had been reported in which the facial nerve was paralyzed during birth. This paralysis occurred in cases of normal delivery without the use of any instruments, and recovery occurred soon after birth.

¹ "Congenital Facial Paralysis," by H. M. Thomas, M.D., *JOURNAL OF NERVOUS AND MENTAL DISEASE*, August, 1898, p. 571.

² Thomas, *loc. cit.*, p. 583.

The facial paralysis in Dr. Langdon's case might have been produced in like manner and recovery have been incomplete. His case was an extremely interesting one and the diagrams showed perfectly the affected muscles. Such a diagram was to Dr. Thomas an entirely new way of showing the paralysis.

Dr. Langdon said that a point of some interest to him was that the patient had been an attendant in asylums and had come in contact with a number of neurologists, as well as other physicians, and nobody seemed to have noticed that he had a facial paralysis of unusual type. The first time Dr. Langdon saw him he noticed that he did not close the eye, but could control the lower face movements pretty well. A feature of therapeutic interest that was not dwelt upon was the consideration of what might be done to relieve the patient. Dr. Langdon thought the ophthalmic fissure might be narrowed so that the eye could be partially closed and thus saved from continual irritation, and he had consulted Dr. Robert Sattler, of Cincinnati, about narrowing the canthus in that way. It would require a narrowing of only about one-eighth inch, and if in addition the lower lid could be fixed a little higher on the cheek, the principal obstacle to closure of the eye would be removed.

200. HYSTERIE TRAUMATIQUE; DOUBLE PIED BOT HYSTERIQUE. AMNESIE RETROANTEROGRADE (Traumatic Hysteria, Hysterical Club-foot, Retroantero-amnesia), A. Sicard and A. Riche (*Presse médicale*, Oct. 15, 1898, No. 35, p. 225).

The patient, a sailor, 19 years of age, without neuropathic diathesis, and without previous history of severe infection, fell a distance of eight meters to the bridge of the vessel. He did not become unconscious. The following day, he attempted to rise, but was unable to do so, and from that time lost memory, not only of the accident, but for the month preceding it, and for the four months succeeding it. Five days after the accident, he was admitted to the hospital, and it was discovered that the lower limbs were crossed, and the feet in position of calcaneo-varus. Three months later, the following descriptive notes were made: Vigorous young man, legs crossed, the feet in a varus position with foot drop, and marked paresis of the legs. When horizontal, it was possible for the patient to support the legs. From time to time, particularly after voluntary movement, there was a violent tremor commencing in the lower limbs, and becoming rapidly generalized with a sensation of epigastric and precordial anguish and of laryngeal constriction. This was not associated with loss of consciousness. There was hypoesthesia with the exception of the feet, where there was complete segmentary anesthesia for touch-, pain-, and temperature-sense. There was also some loss of the muscle-sense; there was concentric retraction of both visual fields. The reflexes were apparently preserved, percussion upon the patellar tendon caused general tremor. A diagnosis of traumatic hysteria was made and a single hypnotic treatment produced complete cure.

SAILER.

A CASE OF CEREBRAL HEMORRHAGIC PACHYMENINGITIS WITH PSEUDO-BULBAR PALSY.

BY CHAS. W. BURR, M.D.,

Professor of Mental and Nervous Diseases in the Medico-Chirurgical College; Physician to the Philadelphia Hospital, and

D. J. MCCARTHY, M.D.,

Associate in Clinical Medicine, William Pepper Clinical Laboratory, University of Pennsylvania; Assistant in Neurology in the Philadelphia Polyclinic.

J. D., male, white, 43 years of age, a peddler, was admitted to the Philadelphia Hospital on April 29th, 1898, on account of epileptiform convulsions. His family history was negative. He had had acute articular rheumatism in 1891, and had used alcohol excessively for years. He denied venereal infection. His present trouble began in July, 1897, with a general convulsion after a debauch. He was unconscious and bit his tongue. Since the onset he has had at least one fit each week. On the day before his admission he had ten attacks, and during the previous week three. He has no distinct aura save momentary dizziness. He falls unconscious immediately. He usually passes a large quantity of urine after an attack. Headache lasting for several hours frequently follows. The attacks are increased in number and made more severe by drinking. For quite a long time he has had constant frontal headaches, at times so severe as to prevent sleep.

Condition on Admission.—He is a thin, pale, old-looking man. His gait is not ataxic, but he staggers a little, apparently from general weakness. There is no ataxia of station. He uses his hands well. Both knee-jerks are increased. Ankle-clonus is absent. The right grip is 45; the left, 32. There is no tremor of the hands, lips or tongue. Sensibility is normal over the entire body, head, trunk and extremities. He localizes touch well. Speech is slow and hesitating. During the first week in the hospital he had four convulsions with unconsciousness. After each fit he slept for about an hour. By May 15th he had become quite stupid, and at the same time very emotional, crying pitifully whenever spoken to. His gait became very uncertain, with a decided tendency to fall towards, or rather sink down on, the left side. He could not stand at

*Read at the twenty-fifth annual meeting of the American Neurological Association, June 14th and 15th, 1899.

The microscopical work is from the William Pepper Clinical Laboratory (Phœbe A. Hearst Foundation).

all with the feet close together. The left grip was very poor, and the arm was held in a paretic attitude. The saliva dribbled. No convulsions occurred between the 9th and 23d of May. The lower part of the face was then noticed to be drawn to the right, especially when crying. By May 24th the weakness on the left side had increased so much that he could not walk. He then began to pass urine and later feces involuntarily. This was due to his mental state, not to palsy of the bladder or rectum. He was dull, emotional, childish, demented. He understood little or nothing that was said, and his speech rapidly became inarticulate. By July 10th he had improved so much that he was able to be up and about the ward. On July 19th he had a convulsion, after which his speech again became bad, and he was once more helpless. During his stay in the hospital a few granular casts were occasionally found in the urine, but never albumin or sugar. He was discharged from the hospital greatly improved, September 6th, 1898, at the request of friends.

He was readmitted November 30th, 1898, complaining of weakness in both legs and difficulty in speaking.

Examination.—He is very poorly nourished. The left side of the face is relaxed, the left corner of the mouth drops, and the right is drawn upwards so that the mouth does not close perfectly on the left side. The tongue when protruded deviates to the left. It is not tremulous. There is great general emaciation, but no local wasting. The left arm is a little stiff. There is motor ataxia in both arms. When in bed he can lift either leg fairly well, and can flex and extend the toes easily, but movement at the ankles and knees is very weak. He cannot stand at all. The knee-jerks are very much increased. Passive lifting of the legs from the bed causes a clonus in them. Patellar clonus is marked on both sides. The legs are so spastic that ankle-clonus if present cannot be obtained. Both the biceps and triceps tendon jerks are present. Tapping the triceps muscle on the right arm causes clonus. The superficial reflexes are all present, and the plantar is especially marked. Sensibility is preserved over the entire body.

On examining the lungs, tactile fremitus is found increased over the right apex and upper lobe. The percussion note is not very unlike on the two sides. The breath sounds are harsh over both apices, especially the right. At the base posteriorly there are occasional coarse râles. The heart and abdominal organs are normal. The patient remained in the hospital only a few weeks, during which time he improved a great deal and was removed at the request of his friends. He was admitted for the third time on February 4, 1899.

Examination.—He is very much emaciated and very weak.

being bed-ridden. He coughs constantly, but cannot expectorate. He swallows with great difficulty. At first he could move his tongue freely, but after a few weeks it became almost immovable. Speech was as a rule inarticulate, but at times he could pronounce a word, clause, or even a short sentence distinctly. The left leg and arm and the lower half of the left side of the face were almost completely paralyzed. The arm and leg were contractured. He was very emotional, crying whenever spoken to, and indeed, whining and weeping for hours at a time. He seemed to understand all that was said to him. Both knee-jerks were much increased. Ankle-clonus could not be obtained, probably on account of the spasm. There were slight bedsores on both hips. Tactile sensibility was preserved. Pain and temperature sense were not tested. He paid no attention to the bladder or rectum.

Dr. de Schweinitz examined the eyes and reported: "Right eye: pupillary reaction normal. Left eye: old synechiæ from iritis. In right eye moderate optic neuritis, apparently of the edematous variety, the retina and nerve-head being cloudy with edema. Veins very full and between arteries normal in size. General absorption of the pigmented retinal epithelium. In left eye conditions similar, but not so marked."

The urine contained neither albumin nor casts.

He was weak mentally. He had quite a number of general convulsions. The temperature was, for the most part, normal, but there were several periods of fever up to 103° , lasting one, two and three days. He died April 29th, 1899.

On the following day a necropsy was held. The calvarium was normal. The dura was adherent to the brain over the entire convex portion of the prefrontal lobe on the left side, and over a circular area two and a half centimeters in diameter, at the apex of the right prefrontal lobe. The dura, where adherent, was very much thickened, and on cross section was seen to have an exudate about one-eighth of an inch in thickness on its outer, and a like exudate slightly thicker on its inner surface. On the inner surface of the dura a fibrinous pseudo-membrane covered the entire convexity on the left side and the frontal and parietal regions on the right. It stripped off easily, leaving the dural surface shining and hard.

The spinal dura from the upper cervical region to the level of the third thoracic segment showed a similar condition. Its greatest thickness was three-sixteenths of an inch. The delicate, fibrinous, pseudo-membrane could be traced to the fifth thoracic segment. The greatest thickening was on the ventral aspect of the cord, and gradually thinned out towards the dorsal aspect. Only the first and second cervical roots were caught in, and

compresed by, the thickened dura, the lower cervical and upper dorsal roots merely passing through, but not being injured by the delicate, soft pseudo-membrane.

Examination of the brain revealed on the second left frontal convolution several small partially caseated, partially organized tuberculous masses, varying in size from a barleycorn to a small cherry. These tubercles were in the cortex, penetrated the sub-cortical tissue slightly, and were situated beneath the dura at its thickest part. The rest of the brain showed no gross lesion. The convolutions were normal in size, the pia stripped readily, although it was somewhat thickened, and a careful microscopic examination of the brain substance, including the internal capsule, showed nothing to account for the motor disturbance. The crus, pons and medulla were very asymmetrical. The left side was distinctly smaller, the motor tract being one-half the size of the right, and lighter in color. Below the crossing of the pyramids, the spinal cord throughout its entire length was so much larger on the left side that we thought of a hemiatrophy secondary to the degeneration of the motor tract of that side. Inequality of the two sides is often seen in normal spinal cords, but cannot usually be traced into the opposite side of the bulb and peduncles. It is striking that the degenerative atrophy, visible to the naked eye, was on the side of the cord opposite to the hemiplegia. The microscopic examination explained this apparent anomaly. It showed the defect in the right side to be of long standing, whereas a recent acute degeneration was present upon the left. The patient had evidently had at some time a right hemiplegia, that had cleared up, leaving symptoms so few and insignificant that they were masked at the time of examination by the preponderance of those affecting the left side, and were considered to be due to general weakness. This was, perhaps, a pardonable error in judgment. That the lesion on the left side was not the so-called tertiary degeneration was proven by the clinical course of the case, and the presence of the lesion as high up as the internal capsule.

Microscopic examination of the central dura proved the external pachymeningitis to consist of two dense layers of newly formed fibrous connective tissue with oval and club-shaped nuclei, separated from each other and the true dura by areolar connective tissue, containing wide blood channels. The layers of the true dura were likewise thickened, with many enlarged and changing nuclei, but preserving perfectly their individuality from the newly formed membrane. The internal pachymeningitis followed the usual type, and presented a highly vascular newly formed fibrous layer, a layer of small, round cells and a mass of organizing fibrin on its free surface. In the ex-

ternal and internal layers evidence of old and recent hemorrhages from vessels with very thin walls was everywhere visible. The internal pachymeningitic membrane was very rich in blood vessels, which consisted mainly of a layer of endothelial cells lining fissures between the layers of fibrous connective tissue. The fibrinous layer contained many free red blood corpuscles and blood vessels in an early stage of formation in its mass. The layer of small, round cells with large, deeply staining nuclei appeared to result from a proliferation of the endothelium of the vessel walls on the free surface of the organized membrane. The marked thickening of the primary layers of the dura, the external pachymeningitis, the round cell infiltration between the layers of the internal pachymeningitis, all point to a true inflammatory process in the membranes of the brain. The condition of the tubercular masses in the cortex, their caseation and partial organization, and the condition of the neighboring cerebral tissue lead us to believe the following was the sequence of events:

An active tuberculosis of the cortex; a secondary inflammation of the dura from the irritation of the tubercular masses or by direct infection, the exudation of fibrin and its organization with the formation of a false membrane which followed the usual pathologic course. It is true that it was impossible to prove the presence of tubercle bacilli in the sections, but since the material was hardened in Müller's fluid, this was hardly to be expected. There were present in the round cell layer a number of bacilli which were of the size and shape of tubercle bacilli, but they did not stain characteristically. Further the tissue examined showed the structure usually found in the false membrane of hemorrhagic pachymeningitis, and was not at all tuberculous. The tubercles of the cortex were typical in structure, and particles of these injected into rabbits gave positive results.

Microscopical examination of the cerebral cortex revealed the following: The usual carmine and nuclear stains stained the tissue as one diffuse mass, the cells and vessels being distinguished only by their pericellular and perivascular spaces. This total lack of differentiation depends, we think, on a chemical change in the tissue, resembling the albumose gelatin exudate described by Echeverria. By staining first in ammonium carmine, then by fuchsin, and overstaining with hemalum the following changes are noted: The subpial layer is very much thickened. There is a diffuse gliar sclerosis characterized by an increase in the number of the gliar cells, with hypertrophy and fat infiltration and a thickening of the gliar network. The smaller ganglion cells have large vesicular

nuclei, their apices point in all directions; a leucocytic infiltration is seen in some and a pathologic pigmentation in all. As to this last condition, though the amount of pigment varies normally, it never in health occupies the entire cell body as was the case here in many cells, or even two-thirds, or one-half

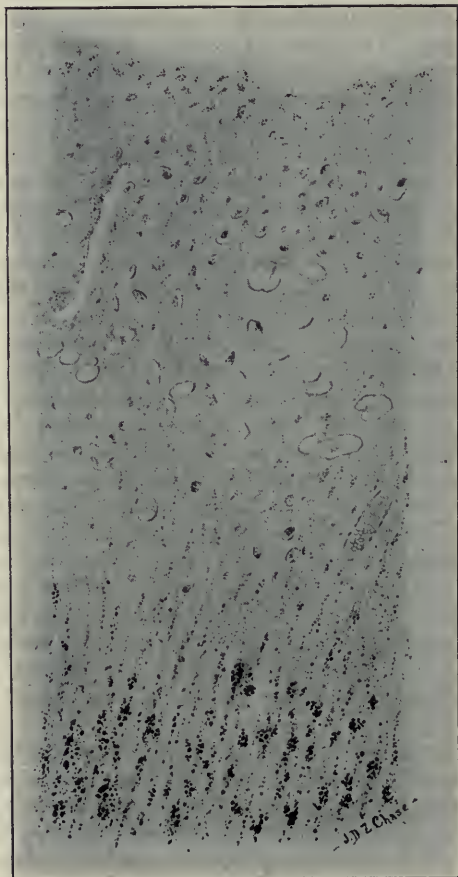


Fig. 1. Section of cortex, motor area. Marchi's stain. The Deiters' cells in the subpial layer show black dots of fat. The hypertrophied glia cells in the white matter are indicated by the accumulations of black dots in the lowermost section of the sketch.

of the cell area, as happened in all. Abnormal pigmentation, however, does not necessarily mean disturbance of function. This pigment staining black with osmic acid was very striking

and extensive. Another pigment staining black with hematoxylin was present in most of the ganglion and glia cells. It is evidently a distinct pigment and of doubtful origin. This double pigmentation was not confined to the cortical ganglion cells, but was found throughout the entire central nervous sys-

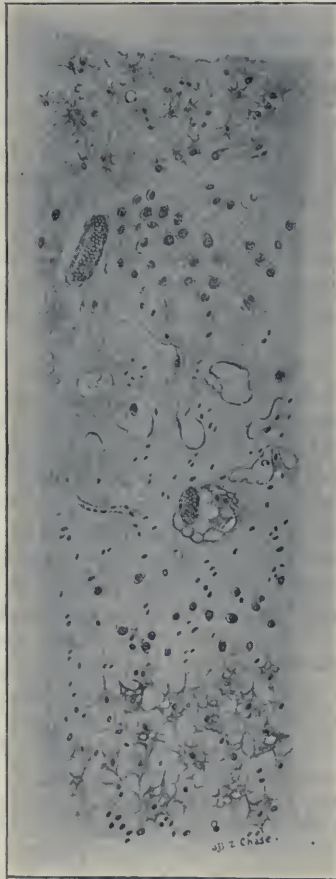


Fig. 11. Section of cortex, motor area. Rosin's stain. The hypertrophy of the subpial glia cells, and of the glia cells in the subcortical white matter, is shown. The cerebral porosis, with a glia cell in one of the spaces near the middle of the picture, is indicated; as is also the scarcity of large pyramidal cells.

tem. The large pyramidal ganglion cells in the deeper layers of the cortex are few in number and show the same changes noted in the smaller pyramidal cells. The so-called leucocytes

in the pericellular spaces and apparently at times in the nerve cells when stained by Ehrlich's triple stain resemble the lymphocytes of the blood.

The tangential fibers in the cortex were fewer than normal, and widely separated in the thickened subpial layer. The terminal projection fibers were also decreased in number, and more widely separated than normal. Association fibers were not observed anywhere in the sections. The gliar meshwork in the entire cortex was much closer and coarser than normal, except in the neighborhood of the blood vessels, where a rarefaction had taken place with the formation of microscopic vacuoles, within which gliar cells and gliar fibers could be observed. (See Figs. I and II.) This is the cerebral porosis of Marinesco and Sérieux. That it is not, as many maintain, simply a dilatation of the perivascular spaces is shown by the presence of glia cells with spider-like processes within the spaces, by the normal caliber of the perivascular spaces of vessels in the immediate neighborhood, and by the peculiar eccentric arrangement of the vacuoles, in many cases extending out from one side of the vessels only. The absence of free red blood corpuscles in the tissue speaks against any marked congestion with dilatation of the capillaries. The whole appearance suggested the absorption of cerebral substance in the presence of a diffuse hypertrophy of the glia—an early stage of a process which might end either in a condition of multiple macroscopic vacuolization of the cortex (Stühlinger and Fürstner), or, as seems more probable, a sclerotic atrophy of the affected convolutions. Examination of the underlying white matter supports this view. The gliar cells formed there a close meshwork, were much hypertrophied, at times four or five times their normal size, and showed in the cell body large fatty granules. This condition probably depends on an absorption by the glia cells of the fatty detritus of the breaking down cerebral substance.

The cerebellum presented the same peculiar staining properties, the same hypertrophy with fat infiltration of the glia, and a rarefaction of the cerebellar substance, modified by the peculiarities of its structure. By the Marchi method, a continuous layer of glia cells, infiltrated with fat, was found at the outer margin of the granular layer. By other stains, a distinct rarefaction in the immediate neighborhood of this layer was shown, presenting the appearance of lacework. In the pons and medulla the same fat infiltration of the cells was seen.

The cells of the spinal cord presented the same double pigmentation above referred to. Instead of the atrophy noted in the cells of the cortex, the cells of the anterior horns were,

if anything, larger than normal. This may have been due to the large accumulation of pigment in the cells. The cell nuclei were centrally located, the processes seemed normal and stained well.

In the white matter Weigert's hematoxylin method showed descending degeneration very marked in the direct pyramidal tract of the left side and the crossed pyramidal tract of the right. It also showed a slight degeneration in the opposite motor tracts. Marchi's method reversed the picture, that is, the most marked degeneration was found on the left side which showed but little change with the Weigert method; the right direct pyramidal tract was also much degenerated and could be traced into the sacral cord. Slight degeneration was shown by the Marchi method in the right crossed pyramidal tract and the left direct tract. In the cervical region a distinct degeneration was noted in Loewenthal's tract. The tracing of these fibers by Marchi's method in and above the bulb had to be omitted, in order to determine whether the case was one of cerebro-bulbar or pseudo-bulbar palsy. The presence of the membrane in the cervical region may have had something to do with the degeneration of Loewenthal's tract. In the upper cervical cord the posterior columns were degenerated, and sections of the posterior roots where they passed through the thickened dura showed the latter to be the cause of the degeneration. In the thoracic and lumbar regions the posterior columns did not show degeneration by the Marchi method; but the carmine and Weigert hematoxylin stain showed sclerosis in Goll's columns. There was marked sclerosis in the left direct and right crossed pyramidal tracts, and a slight sclerosis in the opposite motor tracts.

Macroscopic examination of the thoracic and abdominal viscera by Dr. D. Riesman revealed old and recent tuberculosis, and bilateral gangrene of the lungs; chronic interstitial nephritis; beginning aortic atheroma.

The discovery of the primary cause of the various pathological conditions narrated above resolves itself into a study of the etiology of the hemorrhagic pachymeningitis. Several factors must be considered. Foremost among them is the continued and excessive use of alcohol. It is pretty well proven that it alone may be a sufficient cause. It is not proven, however, that it is always the cause. We have seen more than once a tumor of the cortex, associated with a widespread pachymeningitis, having no other apparent cause than the tumor

itself. The influence of tuberculosis in the development of the condition is of no little importance. That some relation exists between the two diseases has been frequently noted. Of forty-one cases reported by Wiggelsworth, most of which were in the insane, twenty-two had general paralysis, and nine phthisis. In six cases of phthisical insanity reported by Dercum marked changes in the meninges were noted. Fürstner, after calling attention to the presence of delicate pseudo-membranes on the dura of consumptives, says he has so often seen in drunkards, immediately after the first signs of pneumonia, brain symptoms caused by a hemorrhagic pachymeningitis that he is sure the latter was produced by the circulatory disturbance resulting from the infiltration of extensive lung areas. Barthéz and Sanné differentiate intracranial hemorrhage in tuberculous children into medullary hemorrhage more common in acute miliary tuberculosis, and meningeal hemorrhage when the tuberculosis is of the chronic type with glandular enlargements and marked cachexia. Goldscheider and Raymond have reported cases of circumscribed, chronic, tuberculous meningitis of the spinal dura. Indeed a mere glance at the literature shows the frequent association of the two conditions, but whether there is a causal relation between them or not is an unsettled question. There is no reason why the fibroid changes noted in the pleural, pericardial and peritoneal cavities in phthisis should not take place in the intrameningeal spaces. We have already stated the causal relation we believe to have existed between the tuberculous masses of the cortex and the false membrane and dural thickening.

As to the spinal pachymeningitis, in the absence of any local tuberculous growth, it is probable that alcohol was the primary cause:

The diagnosis of hemorrhagic pachymeningitis is always difficult, frequently impossible. It occurs most often in insanity which masks its symptoms, or it is, as in our case, one of many lesions. It may exist for years without appreciable symptoms. It may simulate a variety of conditions, as tuberculous meningitis, progressive paralysis, and brain tumor. Occasionally, however, there is a certain symptom-complex, a particular clinical course from which we may more than guess its

presence. Thus given a man who has used alcohol for years, who is syphilitic or in whom senile changes are marked, the occurrence of an apoplectic or epileptic attack, especially if preceded for some time by headache and vertigo and not presenting symptoms of brain tumor, at least creates a suspicion of hemorrhagic pachymeningitis. The coma of the acute attack is usually not so deep as that in hemorrhage within the brain. There are always symptoms of irritation, contracted pupils, delirium, monoplegic or hemiplegic rigidity. The palsy is not primarily flaccid, as in intracerebral hemorrhage, but spastic from the first.

In days or weeks the patient recovers in greater or less degree mental and physical power, only to lose them again by a second hemorrhage from the partially organized membrane. The attacks recur, and after each the patient is left more palsied and with less mentality, until finally he dies from an apoplectic seizure or from some complicating disease of the lungs or kidneys. The rigidity and increased reflexes at the onset, the absence of cranial nerve involvement, and above all the marked remissions, are important in diagnosis. In cases occurring in the chronic insane in which the hemorrhage is not sufficient to fill up the space caused by the wasting of the convolutions, there often are no noticeable symptoms.

Our patient after a life of drunkenness developed when about forty years old a disease supposed by the committing physician to be late epilepsy. While under observation he began to suffer from intense headache, vomiting, left hemiparesis going on to almost complete palsy, failure of memory and great loss of mental power, and a marked increase in the number of convulsions—a clinical picture which led to the tentative diagnosis of brain tumor. When, however, the exacerbations and remissions became more marked, a slowly forming optic neuritis appeared, palsy of the faucial muscles, ataxia of the arms, difficulty in swallowing, in expectoration, in articulation and in the movements of the tongue, and finally the so-called forced emotional states, so frequently seen in bulbar disease, the difficulties of diagnosis were greatly increased.

Bulbar disturbances are not infrequent in the last stages of

organic insanities, but in this case the ordinary sequence of events was reversed, the dementia following instead of preceding the appearance of the symptoms of gross organic lesions. In form our diagnosis, brain tumor, was correct—tumors were found post mortem, but in fact it was an error, for the whole picture seen at autopsy was unlike that conceived by us while the patient was alive. The exacerbations and remissions were consistent with hemorrhagic pachymeningitis. Inflammatory changes in the optic nerves have also been observed in hemorrhagic pachymeningitis, but not of the kind present in this case. As shown by Fürstner, Griesinger and Oppenheim, there may be an acutely developing choked disk, due as proven by necropsy to an infiltration of blood into the optic sheath. In our case the development was slow, and shortly before death only a marked, cloudy edema of the nerve and retina was observed, probably due to slowly developing and long-continued increased intracranial pressure.

We were thrown off the proper diagnostic track also by the cranial nerve involvement. According to Bullard and others this is very rare in hemorrhagic pachymeningitis. When it does occur the necropsy has always revealed a hemorrhagic infiltration about the base of the brain. This was not present in our case, and it is of interest to determine what was the causation of the bulbar symptoms. That multiple foci of disease in the cerebrum may, though rarely, give rise to symptoms closely resembling the Duchenne type of progressive bulbar paralysis, without any lesion in the bulb, is well established, and that this is the explanation of our case is proven by the microscopic examination. Since serial sections of the bulb show no areas of inflammation or degeneration, we have concluded that a true cerebral form of bulbar paralysis was present, and not the cerebro-bulbar form, in which lesions are found both in cerebrum and medulla.

The cervical pachymeningitis had of course nothing to do with the bulbar symptoms, and caused only the ataxia in the arms. The clinical course was more that of the true cerebral type—a slow and gradual increase in difficulty in swallowing,

progressive slurring of speech until speech became a mere jumble of noises with now and then an explosion into some short commonplace sentence. The forced and spasmodic emotionalism is common in this syndrome, and was very marked in this patient. He would attempt to talk and after a few efforts suddenly break down and begin to cry. Whether this emotion was real, or whether it was only a reflex mimicry of grief, must remain unsolved. This case furnishes a striking example of the pseudo-bulbar syndrome resulting from a purely cortical condition. Pachymeningitis must be a very rare cause. The tubercles, we think, may be left out of consideration as causative factors, because of their position, their size, their long duration before bulbar symptoms appeared, and the fact that they were all in one hemisphere. This last fact is important. The cases of Magnus, Bamberger, Kirchoff, etc., in which unilateral lesions are described as having produced bulbar symptoms, cannot be accepted as accurate in the light of newer methods of microscopic research. In the absence of a thorough microscopic examination, the probability is always in favor of the cerebro-bulbar type of the disease. For a unilateral cerebral lesion to cause the condition would require an anomalous distribution of cortico-bulbar innervation.

The diffuse cortical sclerosis is a possible causative factor, but there is no reason to suppose that in the absence of the pachymeningitis, it would have produced bulbar symptoms. Jolly's case of pseudo-bulbar paralysis with multiple cerebral sclerosis did not resemble ours, clinically or pathologically. In it there were well defined sclerotic patches cutting off certain brain tracts.

The following conclusions may be drawn:

1. Hemorrhagic pachymeningitis should not be forgotten as a possible cause of epileptiform fits beginning in adults.
2. The condition may sometimes at least be inflammatory in origin.
3. Alcohol and tuberculosis are both causes.
4. A pseudo-bulbar palsy may develop in the course of the disease.

ON SENSORIMOTOR PALSIES OF THE MUSCULATURE
OF THE FACE, WITH REMARKS ON THE OCULAR
PALSIES OF THE EARLY STAGES OF TABES.¹

BY JOSEPH FRAENKEL, M.D.,

Physician in charge of the Montefiore Home; Instructor in Nervous
Diseases at the Cornell University Medical School; Neurologist to
the City Hospital.

About one year ago I reported to this society a case of disease of the base of the brain with implication of the fifth nerve, and remarked on that occasion, when speaking about the motility of the face, the following: "The examiner's attention is first arrested by the patient's facial expression. The left side of the face seems contracted; the left-sided temporal and masseter muscles appear atrophied. The left eyelid is drooping. The degree of contraction of the muscles of the left side of the face and the width of the left palpebral fissure are by no means constant; they vary greatly from day to day, and even during one examination. These variations are greater than would correspond to mere physiological changes of the physiognomy, and are confined to the diseased side. For instance, the lid that was drooping at one time is unduly elevated at another time, exposing a part of the upper margin of the sclera. At first it appears as if the right-sided facial nerve were paralyzed, because the folds around the eye and angle of mouth of the left side are much deeper. However, when the patient is put through the usual tests to determine the state of the facial nerve, the right facial nerve is found to be absolutely normal, while in the distribution of the left a phenomenon is noticed, that can best be compared to the ataxia and over-innervation commonly observed in the extremities of tabetic patients. The patient performs all motor functions with the left facial nerve, closing and opening the eyes, showing the teeth, etc., with unnecessary force, with an expenditure of energy that is extravagant compared to the effect desired. At other times these same

¹ Read before the New York Neurological Society, March 7, 1899.

movements are slow, awkward and peculiarly jerky and interrupted."

I have been impressed with the diagnostic significance of the facial expression, as no doubt a great number of us have been. We all have probably evolved for ourselves some clinical rules which frequently enable us to read the disease from the expression of the face. I need only remind you of the Parkinsonian face, of the facial expression of a general paretic or a patient afflicted with bulbar paralysis. Among the tabetic patients a characteristic facial expression is also frequently observed, and I have attempted to analyze this phenomenon since I reported to you the above case.

During these studies good fortune brought another case under my observation which presented many points particularly serviceable in elucidating part of the question to which I propose to invite your discussion to-night. The suggestions received from the first case proved quite helpful in the analysis of the second, to be reported presently, and from this analysis the following points crystallize out for further more thorough investigation:

1. What, if any, is the effect of disease of the fifth nerve upon the motility of the face?
2. Which, if any, function of the fifth nerve is it in particular that is indispensable for perfect motility of the face?
3. The fifth nerve carrying deep sensibility to the ocular musculature as well as to the facial, what—if any—is the effect of disease of the fifth nerve upon the motility of the former?
4. How are we to clinically determine the state of deep sensibility of the facial and ocular musculature?
5. Are not some of the ocular palsies observed in the early stages of tabes sensorimotor paralyses and not primarily motor?

For the solution of these questions the following methods were employed:

1. Study of a case with a rather fortunate grouping of symptoms.
2. Study of the physiological, clinical and anatomical literature at my disposal.
3. Analysis of twenty-two cases of tabes, at present under

my observation, with regard to the relation between disordered function of the facial and oculomotor nerves² and disease of the fifth nerve.

In looking over the histories of other cases of tabes observed during the last six years, I found them insufficient in the particular points under discussion, and as the findings could not be verified now, they were unreliable for the present investigation.

The case referred to is the following:

I. B., forty-two years old, a Roumanian musician, was first seen by me when he was shown in a medical society of this city as a case of *tic douloureux*, two years previous to his admission to the Montefiore Home.

At that time he complained of attacks of severe pain in the left side of the face and in the areas of distribution of the trigeminal nerve; and of the sensation of a heavy helmet on his head.

On October 6, 1898, the patient entered the Montefiore Home and the history obtained after admission to the hospital is as follows:

Born of healthy stock and temperate in his habits, he was well up to his twentieth year. When he was five years of age a hot drink "burned" his throat and the result of this is found to-day in an extensive synechial adhesion between the posterior surface of the soft palate and the anterior surface of the pharyngeal wall. The patient married at the age of eighteen, became father to five healthy children, one of whom died at an early age. His wife never had a miscarriage. He believes that he had a venereal sore when twenty-one years of age. This disease was treated internally and externally during four months and he has not had any further manifestations of the infection since that time. Thirteen years ago an attack of pain and inflammation (probably glaucomatous) destroyed the vision of his left eye.

The present disease began from unknown causes three years ago with attacks of excruciating pain in the face and head, preceded and followed by a sensation of a heavy helmet upon the head and face (*Hutchinsonian mask*). One and a half years ago vision of the right eye began to fail and patient is at present absolutely amaurotic. He has had, and has now, paresthesias here and there in the extremities, particularly in

² The term oculomotor nerves is used here and in other places of this paper in the sense of the general nerve-supply of the external eye-muscles.

the upper ones. No vesical disturbances were noticed at any time. Bowels are costive since onset of the disease and sexual appetite is considerably diminished. During the present illness there was no vomiting. He is at present moaning and groaning day and night, and claims to have unbearable pain and paresthesias in the head and face, particularly on the left side.

He is well built, poorly nourished, and shows few somatic signs of degeneracy. Station and gait are uncertain and ataxic, particularly the station.

The most remarkable symptom is the patient's facial expression. When he is quiet, one would think that there were a paralysis of the lower two branches of the left facial nerve. When he is made to laugh or cry, or when he does so in response to autochthonous psychical stimulation, only the upper branches of the left and the lower branches of the right facial nerve go into play. This gives to the face an indescribably contorted expression. These movements show, moreover, a considerable over-innervation; the contractions of the left frontalis and orbicularis oculi and of the right buccal musculature are executed with a good deal of force and vigor, quite disproportionate and not alike on all occasions. Apparently as a result of this phenomenon the skin around the left ocular and right oral aperture is thrown into deep folds while the rest of the face continues in a smooth, masklike expression. This expression is vastly different from the Parkinsonian facial immobility; the stiffness of the latter is replaced here by an atonic relaxation of the facial musculature. The left palpebral fissure is of varying width; at a given moment the fissure is smaller than normal; at another the upper lid is unduly elevated, exposing the upper scleral margin.

The head is in general tender to percussion, perhaps a trifle more so in the occipital region. The head is moved freely in all directions, but passive excursions of the head appear to be painful, to judge from the change in the facial expression. On such an occasion the lower branches of left facial, so far apparently paralyzed, bring the muscles about the angle of mouth into vigorous contraction and partly equalize the mentioned facial asymmetry.

Sense of smell and sense of taste are considerably impaired on both sides.

The patient is completely amaurotic; the ophthalmoscope reveals a complete bilateral optic atrophy. The ocular axes are divergent. Left pupil is key-hole shaped and obstructed by a grayish mass. Right pupil is well shaped and reacts to light. When the patient is made to look at the point of his nose and then into the distance the right pupil dilates in the first instance

and contracts in the second. The excursions of the right eyeball are normal. The muscles of left eyeball seem at first parietic, the excursions being very much less prompt and below the physiological limits. In lateral positions nystagmiform twitchings are observed. When patient is made to repeat frequently the task of bringing the left eye into different positions the promptness and vigor of the contractions improve gradually and are finally fairly good, though not as good as on the other side, but decidedly better than they were at first. The nystagmiform tremor of the eye muscles is also considerably lessened after such exercise.

On a later occasion the same exercise has to be repeated; the improvement in the motility of the eye muscles of the left eye not being permanent. The extent of the limitation is equally unlike on all occasions as are also the contortions of the face and the width of the left palpebral fissure.

To all appearances not one of the external eye-muscles of left side is found actually paralyzed, but all the movements of this eye are unequally balanced and co-ordinated; exaggerated or diminished on different occasions and improve considerably under exercise.

Active innervation of facial musculature is similarly undisturbed in the common sense of the term; all muscles respond to intentional innervation, but with varying force on different occasions, particularly on left side. The temporal and masseter muscles of left side are atrophied, and when the inferior maxilla is moved downward it deviates toward the right side.

Soft palate is attached to pharynx, is immovable, and this impairs considerably the swallowing and speech of patient. The speech is, however, not only nasal but otherwise dysarthric.

Tongue is protruded straight, freely movable in all directions, of good color and shows no tremor or atrophies. The patient lost all his teeth without pain during the present illness.

Extensive disturbances of the sensibility in the distribution of the trigeminal nerves, particularly of the left, are found, and comprise all qualities of sensibility including the deep sensibility. I shall speak later in regard to the method of ascertaining the latter.

The upper extremities show good motor and co-ordinating power and the deep reflexes are present. Undeniable hypotonia exists.

No paralyses or atrophies are found on the trunk. Scattered areas of anesthesia, analgesia and delayed conduction are found. Skin reflexes are present.

On lower extremities slight ataxia and marked hypotonia

are observed; knee- and Achilles-jerks are absent; plantar reflexes are lively. Considerable disturbances of tactile, pain- and temperature-sense exist on the feet and lower third of the legs; deep sensibility apparently intact. Vegetative organs normal.

The final diagnosis of the case reported, *tabes* or *tumor* on or near the cerebellum is somewhat doubtful. All evidence points rather towards the diagnosis of *tabes*. There is unmistakably extensive disease of the fifth nerve, particularly on the left side, and the symptoms dwelled upon at length are clearly dependent upon the trigeminal disease. To recapitulate these symptoms briefly, we could say that there is motor impairment of the facial and ocular musculature, particularly of the left side. The motor defects are of varying intensity and extensity, and are defects of the co-ordination rather than of the motor power proper, as is shown by the fact that the extent of the paralysis varies on different occasions; and particularly concerning the eye-muscles by the fact that exercise overcomes a part of it. Disease of the fifth nerve is evidenced by the disordered sensibility in the distribution of the fifth nerve, particularly on the left side, by the atrophies of the temporal and masseter muscles of the same side, and finally by the loss of the teeth. The tactile, pain and temperature anesthetics were mostly marked in the distribution of the first branches of the trigeminal and were varying; the deep sensibility, however, was permanently and considerably injured.

Before entering upon the further discussion and before attempting to answer the questions formulated at the outset, a physiological preface and short review of the literature seems apropos.

For various reasons it is helpful and instructive to establish an analogy between the cranial and spinal nerves, and further between the cranial and spinal anterior and posterior roots. This analogy may not be anatomically exact, but is quite probable from the embryological and physiological standpoint. So we may compare the trigeminal nerve to a posterior root, and look upon its motor portion, the facial, the oculomotor and trochlearis, as the anterior partner. Excepting, perhaps, the vegetative functions, particularly the motility of the heart where

an automatic apparatus is believed to regulate the motor variations of this organ, it is gradually developing into an established fact for the cerebrospinal functions, that all centrifugal nerve impulses are not autochthonous and independent. In other words, the belief is forced upon us that all centrifugal nerve functions—motility, secretion and trophic influences—are conditioned and regulated by centripetal or sensory impulses. This is well supported by experimental and pathological experience. We all know that section of the posterior roots leads not only to disturbance of the sensibility, but of motility as well. The latter is, of course, not a paralysis in the current sense of the term; the motor functions are altered after section of the posterior roots in kind only; the movements lose their purposefulness and proper adjustment. So far the only tenable explanation of ataxia seems the Leyden-Goldscheider theory, and the ataxia without sensory disturbances called for by Goldscheider is yet to be shown.

Marinesco has furthermore proven by anatomical investigation that abolition of the nerve impulses traveling constantly along centripetal pathways finally derange the structure of the motor apparatuses with which these pathways are anatomically and physiologically connected. Oppenheim and Schlesinger finally succeeded in finding the intermediate centripetal pathways along which the ascending degeneration is supposed to travel.

The influence of sensibility upon the motility is discussed with fair interest in physiological literature. In the neurological text-books and pamphlets this subject is treated rather briefly, and particularly the question concerning the changes of the facial and ocular motility as influenced by sensibility, most text-books pass tacitly.

Exner defines sensomobility as the ability to perform motor functions in so far as they are guided, influenced or regulated by centripetal nerve impressions. Disturbances of the sensibility, according to this author, lead to various alterations of the motility, depending upon the extent and kind of the former as well as the latter. These motor disturbances are produced by the suppression of one or more of the sensory regulations. As

yet there are no distinct differences between the various forms of sensorimotor palsies.

It was known already to Bell and Magendie that the state of sensibility of the face greatly influenced the motility. Experiments led these authors to the conclusion that cutting the fifth or seventh nerve caused the same result, *i. e.*, inability of the animal to grasp its food, but for different reasons: in the first instance owing to the disordered sensation, in the second to the disordered motion. Here we find explanation for the fact that Bell frequently expressed the opinion that the fifth nerve is a motor nerve in some respects. Disturbances of the sensibility of the tongue do not often demonstrably cripple the mobility. The fact that the lingual as well as facial musculature is most of the time bilaterally innervated accounts for the rarity of clearly demonstrable motor disorder when the sensibility is interfered with. In functions that are unilaterally represented, as for instance the function of speech, disturbances are frequently seen. Dysarthria and thickness of speech is frequently seen when the sensibility of one-half of the tongue has suffered. A good illustration of the interdependence between sensation and motion is the experiment of Exner in which he succeeded in producing a paralysis of the vocal cords after section of the laryngeus superior.

Landois, when speaking about this subject, states that sometimes section of sensory fibers is followed by total abolition of the motor function.

In Baker and Harris' "Handbook of Physiology," ed. 1885, the following is found: "In relation to muscular movements the branches of the greater portion of the fifth nerve exercise a manifold influence on the movements of the muscles of the head and face and other parts in which they are distributed. They do so in the first place by providing the muscles themselves with that sensibility without which the mind, being unconscious of their position and state, cannot voluntarily exercise them. It is probably for conferring this sensibility on the muscles that the branches of the fifth communicate so frequently with those of the facial and hypoglossal and the nerves of the muscles of the eye."

On the other hand, Schiff feels justified, from his experi-

ments, in expressing his view to the effect that after section of the fifth nerve on one side, the movements of the face are equally well executed on both sides, and that only as a matter of course the animals, being robbed of the sensation in one-half of the face, will contract these muscles less frequently. He therefore thinks it erroneous to state that cutting the fifth nerve interferes with the motility of face or eyes.

This chapter is apparently beginning to absorb more neurological interest to judge from the controversy between Korniloff and Bickel in the XII and XIII volumes of the *Deutsche Zeitschrift für Nervenheilkunde*. Korniloff exhibited at the last meeting of the south German neurologists and psychiatrists a series of animals with severed posterior roots. In all of them there was more or less extensive disorder of the motility, amounting in some to apparent total paralysis. Korniloff further endeavors to find the kind of sensibility that may be incupated; but the facts so far collected do not permit a final conclusion. Bickel opposes these deductions and claims that an undisturbed sensibility is not indispensable for the soundness of the motility. It seems to me that Bickel is taking the word paralysis in the usual sense, meaning actual motor palsy, and that he would be willing to admit that sensory disorder changes the motility where co-ordination and reflex motion are concerned.

Manifestly the sensorimotor inter-relation has commanded considerable physiological thought since the attention was first drawn to it by Bell and Magendie, and it is evident that all physiologists, excepting Schiff, agree that this interdependence is a considerable one.

As stated already this subject has received less thorough consideration at the hands of the neurologists. Gowers believes that "the movements of the head, face, tongue and eyes escape the characteristic derangement of tabes," although he states, when speaking about the symptomatology of disease of the fifth nerve, that "the muscles of the face are insensitive but not weakened, although the movements of the face have been observed to be a little slower than normally, apparently from defective sensation."

In Dana's text-book the relation between sensation and motion in the face is not discussed. The author remarks, however, that "a slight drooping of one or both lids is not infrequent in the early stages of tabes. It begins early and progresses slightly up to the later stages of the disease. It is due to a paralysis of the sympathetic nerve fibers of the lid."

Dercum's text-book contains from the pen of Peterson the statement that the muscles of the trunk are also ataxic, but the inco-ordination does not affect the movements of the face, tongue, eyes or head. In the same volume Herter does not discuss the influence of disease of the fifth nerve upon the motility of the face.

Rosenthal's "Treatise on Nervous Diseases," translated by Putzel, contains a few very suggestive lines. He states: "The trigeminal nerve is sometimes implicated in ataxia. In a case observed by Duchenne, double paralysis of the fifth pair of nerves occurred with paralysis of the left motor oculi communis. In four of my cases paralysis of the trigeminal and motor oculi communis occurred twice, paralysis of the latter nerve and of the facial and trigeminal developed once. * * * The facial nerve is rarely involved, but certain muscles of the face manifest diminished tonus."

In the newer text-books of Oppenheim and Schultze the sensorimotor inter-relation is very briefly alluded to.

The facial nerve is unanimously reported to be very rarely affected in tabes, and it is perhaps probable that in the cases in which such affection was observed it was not a genuine facial palsy, but a disordered facial motility of the type discussed.

The question is of considerable neurological interest in regard to the pathogenesis of tabes. We frequently hear the objection raised when we attempt to look upon tabes as a disease of the sensory neurons, that the facial and oculomotor nerves are not sensory nerves, and both are involved in the tabetic process, the latter vastly more frequently than the former.

The ocular palsies are according to all authors very frequent and very early symptoms of tabes. With equal uniformity these ocular palsies are characterized as dissociated, slight and transient. All these characteristics are not fully compati-

ble with some of our cardinal conceptions about disease of the motor neuron, and the nature of these ocular palsies is therefore still *sub judice*.

Not much light has as yet been thrown on this question by histological and anatomical investigation. Negative post-mortem findings are just as often reported as degenerative changes in the nucleus, peripheral fibers, or in the muscles. de Schweinitz writes in Dercum's handbook concerning this question thus: "We are uncertain as to the exact cause of these palsies, some being explained as nuclear, others as the result of a descending neuritis, and still others as due to a chronic ependymitis. One of the characteristics of tabetic paralysis is its transitory nature in many instances, and by this peculiarity it may often be differentiated from the lesions of the nerve trunks themselves."

Thus the matter stands at present—quite remote from a satisfactory final solution and hence the justification for the attempt to view the question from some uniform standpoint.

All that seems so far established about the tabetic ocular palsies is the following:

1. They are frequently sudden in onset, of short duration, and disappear just as suddenly without treatment; or they are of longer duration and then disappear mostly under specific medication, or they are permanent.

2. They are frequently dissociated (paralysis of the levator or rectus internus alone), or the paralysis affects all muscles or muscle groups supplied by a given nerve.

3. The extent of the paralysis is sometimes, as I have observed in the case reported and in two others included in the appended table, varying in short intervals and frequently diminished after exercise.

4. Their anatomical substratum is not one and the same; negative findings are just as often reported as lesions of the respective nuclei, nerve trunks or muscles.

Theoretically, there are only two possibilities: first, these ocular palsies are either pathological occurrences of another nature than the general disease they are implanted upon; they share with the general disease the common etiology only, *i. e.* the syphilitic infection, and are pathologically primary or sec-

ondary nerve inflammations in distinction from the pathological lesion of the general disease—a primary degeneration of the sensory neuron. Or, secondly, these ocular palsies are a part of the general disease, *i. e.*, a primary degeneration of the sensory neuron. The degeneration aided by so far unknown circumstances affects in some cases the sensory neuron of the cephalic extremity, and leads to first functional and later structural changes of the corresponding motor neuron—in this instance the oculomotor nuclei, nerve trunks or muscles.

It seems that both mentioned types of ocular palsies are met with in tabes and that the fact of their not being sufficiently kept asunder is obstructing the way to a clearer conception of their pathogenetic relationship to the general disease. The first type is oftener seen in those cases that are rather forms of multiple cerebrospinal syphilis of the tabetic type, and the second in the class of cases that lean more towards the picture of a genuine tabes. In the first instance they may therefore be looked upon as syphilitic ocular palsies occurring in a case of multiple cerebrospinal syphilis of the tabetic type; and in the second instance they are essentially the same phenomena as the motor phenomena in the extremities of tabetics.

It is for the second type of ocular palsies that the view of a primary sensory origin seems to me justified.

These palsies are in their earlier stages simply due to the loss of tonus which is mediated through disease of the sensory neuron, and later on when owing to the long duration or the severity of the process in the sensory neuron the motor neuron has undergone secondary changes, the palsies are lasting, more extensive and their immediate cause found anatomically in degeneration of the nucleus, nerve trunk or muscle. This view is not only quite compatible with the clinical and anatomical characteristics of this type of palsies outlined above, but receives some further support from the fact that it permits a uniform pathogenetic conception of the general disease, and is finally gaining probability from the results of the analysis of the twenty-two cases appended.

The analysis of these cases was undertaken with the aim to ascertain the relation between defective function of the facial and ocular musculature and disease of the fifth nerve. I am

indebted to Dr. Abrahamson for having kindly tabulated for me the results and made the examinations in the following way:

Every patient was examined as to the previous or present existence of trigeminal or oculomotor symptoms. The previous existence of oculomotor symptoms was assumed from a history of double vision or squint; and previous trigeminal symptoms from paresthesias or pain in the face, from masticatory spasms, eye inflammations or loss of teeth. In the examination of the present condition we proceeded in the following way: 1, examination of the ocular excursions; 2, examination of the trigeminal nerve; 3, examination of deep sensibility of face and eyes. The deep sensibility of the facial musculature was ascertained by means of the faradic current, and the patient was interrogated as to his knowledge of the degree of a given contraction. These findings are frequently unsatisfactory. I derived more satisfaction from the procedure of ascertaining the deep sensibility of the eyelids. The most satisfactory results were obtained from two patients, the one reported in this paper and another included in the table, both of whom are totally amaurotic.

When the lid is carefully and gently lifted up by the eyelashes in the external canthus, the patient either distinctly states that the lid is moved upward or downward when the deep sensibility is intact; otherwise he is just as misinformed about the position of his eyelid and looses it somewhere on the eye, as the tabetic patient looses his limbs in the bed. With unimpaired vision the test is less accurate, but still reliable if the examination is made with caution and patience. Under such circumstances the patient is made to look fixedly at an object in the center of the visual field, while he is directed to answer quickly, without making searching movements with the lid after it was lifted or lowered, what the examiner did with it.

In order to determine to what extent the defect of the deep sensibility of the lids is caused by the concomitant amaurosis, the same examinations were made with a large number of other patients. In this type of cases the passive excursions of the lids are acutely and promptly perceived, but it must be stated that in amaurotic persons the eyelids are frequently found

drooping, although the levator palpebræ superioris is otherwise quite intact. In this instance the semi-ptosis is also an expression of a primary sensory defect—the disease of the optic nerve. The endeavor was further made in case evidence of disease of the ocular muscles was obtained, to ascertain to what extent exercise would overcome the existing defect.

Finally attention was paid during the examination to the state of the facial and trigeminal nerves in their lower branches. The detailed results of this examination are embodied in the appended tables and I shall give here only a condensed synopsis of same.

1. Out of the 22 cases examined, 10 give distinct evidences of a previous venereal infection; in 4 cases an infection may be assumed from the accompanying symptoms, and in 8 no reliable information could be gotten.

2. Eight out of the 22 cases give a history of derangement of the ocular musculature previous to the date of the examination, and in 14 no such history was obtainable. Thus it would appear that ocular palsies occur in 36.4 per cent. of the cases of tabes.

3. Two of the 8 cases show a distinct history of previous syphilitic infection; in 2 a venereal infection is probable, and in 4 it is apparently absent.

4. All 8 cases showed evidence of disease of the fifth nerve.

5. Amongst the 14 cases that were apparently free from disease of the muscular apparatus of the eye, 8 had a venereal infection, 4 denied the same, and in 2 it was probable.

6. In 6 out of the 14 cases a history of derangement of the fifth nerve was obtainable. These are cases 1, 4, 10, 12, 15, 17, and it is interesting to note that in cases 1 and 4, optic atrophy was the earliest symptom, so that an eventual disorder of the ocular musculature may have escaped being noticed by the patient. This is the more probable, as both these cases show at present both disease of the muscular apparatus of the eye and of the fifth nerve. In the other 4 cases the only evidence of previous disease of the fifth nerve was surmised from the statement of a painless loss of teeth, *a priori* not very likely to be thought of as conditioning disorder of the motility of the ocular musculature.

7. Six out of the 22 cases show at present evidence of disease of the ocular muscles. Two cases give a distinct history of syphilis, in one the infection is probable, and in three apparently absent.

8. In all these 6 cases there is evidence of extensive disease of the fifth nerve, and in 5 the deep sensibility of the eyelids was found markedly disturbed.

9. Sixteen cases show no disorder of the eye-muscles at present, and in 9 of them disease of the fifth nerve was demonstrated, but only in 3 the deep sensibility of the eyelids was defective.

10. It is very remarkable that 15 of the 22 cases show more or less marked ptosis, or better said, more or less considerable narrowing of the palpebral fissure.

11. In 6 out of the 22 cases derangement of the facial musculature was found, and in all these corresponding derangement of the fifth nerve.

The reasons stated above and the results of the table corroborate the belief in the possibility that some of the ocular palsies of tabes may not be purely motor, but sensorimotor paralyzes, conditioned through primary disease of the sensory neuron and not due to primary disease of the motor neuron.

Further support is lent to this conception by the results of experimental and anatomical investigations made by Marinresco, Oppenheim, Schlesinger and Grabover, who traced disease of motor cells or fibers to primary disease of the corresponding sensory neurons.

Now we may attempt to answer the questions formulated at the outset:

1. Disease of the fifth nerve interferes with the motility of the face.

2. As yet no function of the trigeminal in particular can be ascertained which could justly be supposed to be the cause of the impaired motility; the kinesthetic sensibility when cautiously and patiently examined was found more frequently and more extensively disordered than the other functions.

3. The effect of disease of the fifth nerve upon the motility of the eyes seems to be a similar one.

4. All the means at present at our command for examining

Syphilitic infection.	Were there any symptoms of disease of		Present status.		Facial innervation.	Remarks.
	Eye-muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.		
No. 1—Denied. Two miscarriages.	Apparently not.	Paresthesias over forehead, both eyes and cheeks. Loss of teeth—painless.	Convergent strabismus. Weakness of both external recti, especially right, both superior recti, marked ptosis on both eyes, especially left.	Sensibility: tactile: spots of anesthesia on forehead, hypesthesia on tip of nose. Pain: large areas of analgesia especially left face; also over the right face, excepting portions of cheeks and forehead where also some hypalgesia. Temperature: normal. Deep: disturbed in both eye-lids.	Normal.	The early optic atrophy probably prevented the recognition and perception of ocular palsies by the patient, even if they had existed at the earlier stages of the disease. The convergent strabismus is extreme, but after exercise patient succeeds in bringing the eye-balls fairly well out to right and left side—after exercise the paralysis disappears almost entirely.

Syphilitic Infection.	Were there any symptoms of disease of		Present status.		Facial innervation.	Remarks.
	Eye-muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.		
No. 2—Denied.		<p>Paresthesias over forehead, eyes and cheeks. With the onset of disease had without assignable cause a conjunctivitis—not severe—for 8 days. Loss of all teeth in upper and very many in lower jaw, some with, others without pain.</p>	<p>Convergent strabismus; marked weakness of right external rectus, both superior recti. In extreme position movements of eyeballs are accompanied by a tremor of eyebrows. Partial ptosis of both upper lids.</p>	<p>Atrophy of temporal and masseter muscles of left side. Sensibility: tactile: on forehead spots of anesthesia and delayed perception. Pain: analgesia over forehead, especially right; over both sides of the face large areas of hypalgesia. Temperature: on forehead, both sides, anesthesia; over face spots of anesthesia, delayed and perverted. Deep: disturbed on both upper lids.</p>	<p>Movement of right-side of face not so exaggerated as left, but on both sides somewhat ataxic.</p>	<p>Exercise improves considerably the ocular excursions.</p>
No. 3—Denied.	<p>Diplopia since 4-5 years upon looking to the right, only occasionally and of short duration.</p>	<p>Shooting pain in head. Lost 4 teeth since onset of disease, began to break down painlessly.</p>	<p>Some weakness of right externus. Both palpebral fissures diminished in size.</p>	<p>Sensibility: tactile: normal. Pain: areas of hypalgesia in center of forehead; over right side of face areas of hyperalgesia. Temperature and deep: normal.</p>	<p>Normal.</p>	

Were there any symptoms of disease of	Present status.		Facial innervation.	Remarks.
	Eye-muscles?	Fifth nerve?		
Syphilitic infection.				
No. 4—Venereal sore 18 years ago.	Never diplopia.	Loss of teeth in upper jaw.	<p>Eye-muscles. Ocular movements show nystagmoid oscillations of the eye-ball on looking to right or left. Some weakness of the muscles, especially both external and internal recti. Partial ptosis of both eyelids—palpebral aperture much smaller.</p> <p>Fifth nerve. Sensibility: tactile: normal. Pain: normal. Temperature: normal. Deep: disturbed in the eyelids, normal in lips.</p>	Disease began with difficulty of vision and nystagmia. As in case 1, the fact of early abolition of vision may have masked an eventual disorder of eye muscles.
No. 5—Venereal sore 30 years ago.	Frequent and transient attacks of diplopia.	<p>Formation of forehead, both eyelids, cheeks along both sides of the nose; suffered very frequently from hordeolum. Three years ago lost without pain all teeth in upper jaw except 4, dropped out while eating; in lower jaw all molars and bicuspsids except one.</p>	<p>Some atrophy of left temporal. Sensibility: tactile: normal. Pain: areas of hyperalgia on both sides of forehead and nose. Temperature: disturbed. Deep: on upper eyelids, slightly disturbed, lips more involved.</p> <p>Marked over-winking of left side of face; marked winking of both upper lids in tight closure of eyes.</p>	Test for deep sensibility somewhat difficult.

Syphilitic Infection.	Were there any symptoms of disease of		Present status.		Facial innervation	Remarks.
	Eye-muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.		
No. 6—Venereal sore 37 years ago.	Three years ago diplopia, coming suddenly, lasting 3 months, disappeared completely.	Often double-sided neuralgia, no paresthesia, no masticatory spasm. Loss of teeth since onset of disease.	Palpebral fissures a trifle smaller.	Sensibility: tactile: normal. Pain: analgesia of both ears and right temple. Temperature: analgesia over ears; over right temple diminished and perverted. Deep: normal.	Normal.	Some defect of left facial.
	No. 7—Denied.	Paresthesia over left eye and cheek.	Ocular movements normal. Left ocular aperture smaller than right.	Temporal atrophy left; states that when he had diplopia, the sinking in of left temple took place. Sensibility: tactile: spots of anesthesia in left frontal region. Pain: large areas of analgesia over left side, area also over right forehead; extensive areas of hypalgesia on both sides. Temperature: disturbed over left side of face, where anesthesia, and perverted. Deep: markedly disturbed on left eyelid.		

Were there any symptoms of disease of	Present status.		Facial innervation.	Remarks.
	Eye-muscles?	Fifth nerve?		
No. 8 — Venereal sore 26 years ago.	Never diplopia.	None.	<p>Ocular movements exaggerated—nystagmoid movements. Palpebral fissures less than normal and smaller on left than right.</p> <p>Sensibility: tactile: normal.</p> <p>Pain: hypalgesia on forehead.</p> <p>Temperature: normal.</p> <p>Deep: normal.</p>	
No. 9 — Venereal sore 20 years ago.	Never diplopia.	None.	<p>Some nystagmoid movements on looking to right. Palpebral fissures smaller.</p> <p>Sensibility: tactile: normal.</p> <p>Pain: hypalgesia on left side.</p> <p>Temperature: normal.</p> <p>Deep: somewhat disturbed on both eyes, especially left.</p>	Double optic atrophy.
No. 10 — Denied. Symmetrical suspicious looking scars.	Never diplopia.	Loss of teeth without pain.	<p>Ocular movements normal. Palpebral aperture diminished.</p> <p>Sensibility: tactile: normal.</p> <p>Pain: large areas of analgesia and hypalgesia on both sides of face.</p> <p>Temperature: anesthesia over lower portion of right face, areas of diminished and delayed sense.</p> <p>Deep: disturbed on right upper eyelid.</p>	Normal.

SYPHILITIC INFECTION.	Were there any symptoms of disease of		Present status.		Facial In-nervation.	Remarks.
	Eye-muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.		
No. 11—Venereal sore 23 years ago.	No diplopia.	None.	Ocular movements normal. Tremor of lids. Right palpebral aperture wider than left. Partial ptosis, especially left.	Sensitivity: tactile: normal. Pain: analgesia over left lower jaw laterally, hypalgesia over cheeks, etc. Temperature: left cheek same area anaesthesia, areas of hyperesthesia and hypesthesia. Deep: normal.	Fairly good.	
No. 12—Venereal sore 33 years ago.	Never diplopia.	Loss of teeth.	Ocular movements normal.	Sensitivity: tactile: normal. Pain: both sides of face analgesia. Temperature: somewhat disturbed on forehead. Deep: normal.	Right better than left; upon opening mouth wide left angle lower.	
No. 13—Venereal sore 18 years ago.	Never diplopia.	None.	Ocular movements normal.	Sensitivity: tactile: normal. Pain: area of hypalgesia on central forehead. Temperature: normal. Deep: normal.	Normal.	

Syphilitic infection.	Were there any symptoms of disease of		Present status.		Facial innervation.	Remarks.
	Eye-muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.		
No. 14—Venereal sore 12 years ago.	Never diplopia.	Since onset of disease suffers from hordeolum. Teeth excellent.	Ocular movements rather exaggerated, extreme positions are accompanied by tremor of eyebrows. Space between eyelids normal.	Sensitivity: tactile: marked hyperesthesia over left side of face especially cheeks. Pain: hyperalgesia on left; hypalgesia on right side. Temperature: normal. Deep: normal.	Both sides exaggerated.	
No. 15—11 years ago.	Never diplopia.	Teeth decayed and fell out soon after onset of present disease.	Excursions of eyeballs exaggerated though eyes are quite prominent; some tremor of eyebrow on extreme fixation.	Sensitivity: tactile: normal. Deep: normal. Temperature: normal. Deep: of upper eyelid normal, of the mouth disturbed especially on upper lip.	Left forehead more wrinkled: mouth drawn more on left side and in opening of left angle of mouth and left side of jaw.	
No. 16—Denied.	No diplopia, no squint.	Years ago had neuralgia on both sides of forehead. One tooth in upper jaw, 7 in lower jaw.	Ocular movements normal.	Sensitivity: all qualities: normal.	Normal.	

Syphilitic infection.	Were there any symptoms of disease of		Present status.		Facial In-nervation.	Remarks.
	Eye-muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.		
No. 17—None.	No diplopia, no strabismus.	Loss of teeth.	Ocular movement normal. Narrowing palpebral aperture.	Sensitivity: tactile: normal, Pain: areas of analgesia and hypalgesia on both sides of face. Temperature: normal, Deep: normal.	Normal.	
No. 18—Denied.	No diplopia.	None.	Ocular movements normal. Tremor of eyelids.	Partial atrophy of right temporal. Sensitivity: tactile: Pain: normal, Temperature: hypalgesia to cold on both sides, less so to heat. Deep: normal.	Normal.	Completely amaurotic.
No. 19—Denied.	No diplopia.	None.	Palpebral aperture narrow, especially on right eye.	Sensitivity: all qualities: normal.	Normal.	
No. 20—Denied One miscarriage.	Says that at times when feeling weak, sees double.	Some paresthesias of face. Loss of teeth since onset of disease.	Ocular movements good.	Sensitivity: tactile and pain: Normal. Temperature: large areas of anesthesia and perverted sensibility over both sides. Deep: normal.	Normal.	

Syphilitic infection.	Were there any symptoms of disease of		Present status.		Facial innervation.	Remarks.
	Eye-muscles?	Fifth nerve?	Eye-muscles.	Fifth nerve.		
No. 21.—Denied. One miscarriage.	Diplopia began 12 years ago and exists off and on ever since. Twitching of eye-muscles.	Very often neuralgic attacks, beginning in lower jaw like a tooth-ache; paresthesias of the forehead.	Ocular movements normal. Partial ptosis on both eyes.	Sensibility: tactile: normal. Pain: hyperalgesia on right cheek. Temperature: normal. Deep: normal.	Normal.	
No. 22.—Denied.	Diplopia frequently.	Paresthesias of face. Loss of teeth without pain.	Excursions of eyeballs exaggerated, no evidences of paralysis, brings it out with ease beyond normal, some nystagmoid movements. Palpebral fissures unequal; semi-ptosis covers almost upper half of cornea, varies now and then; strabotic attitudes of eyes.	Evidences of slight atrophy of masticatory muscles. Sensibility: tactile: normal. Pain: delayed conduction, much analgesia — hyperalgesia — hyperalgesia chiefly on left side. Temperature: Deep: disturbed in eyelids.	Ataxic.	

the deep sensibility of the facial and ocular musculature are not very exact. It is particularly difficult to ascertain slight disorder. The procedure described above gives a fair impression of the deep sensibility of the levator palpebræ, and no method is apparently known for determining the kinesthetic sensibility of the other external eye-muscles.

5. Some of the ocular palsies of tabes are probably sensorimotor palsies.

201. LA LOCALISATION MOTRICE MÉDULLAIRE (The Spinal Motor Localization.) Van Gehuchten and Nelis (Journal de Neurologie, Aug. 5, 1899, No. 16, p. 301).

The experiments of Marinesco, according to Van Gehuchten and Nelis, show that the peripheral nerves arising in the different plexuses do not have a definite nuclear localization in the spinal cord; the spinal motor localization is not "nerveuse," *i. e.*, not determined by the nerves. Sano believed that each muscle of the body has a distinct nucleus in the gray substance of the spinal cord. The investigations of Sano, Kohnstamm and Marinesco show that the group of cells in connection with the diaphragm form a distinct column in the center of the anterior horn, but it does not follow that muscles of the extremities have a similar representation in the spinal cord. The motor localization is therefore not "muscular," but is "segmental," *i. e.*, the different groups of cells are in relation with muscles of a segment of a limb. Van Gehuchten and De Buck showed in an earlier paper that amputation of the leg at the knee caused chromatolysis in two groups of cells in the postero-lateral part of the anterior horn; one group extending from the upper part of the fifth lumbar segment to the lower part of the third sacral segment; the other from the upper part of the second sacral segment to the lower part of the fourth sacral segment. Van Gehuchten and Melis found in a case of amputation of both feet that the chromatolysis was limited to the lower group, from which they conclude that all the muscles of the foot are represented in this group, and those of the leg in the upper group. In the cervical cord of man we should expect to find three distinct cellular groups; one for the hand, one for the forearm, and one for the upper arm. We should find three groups also in the lumbosacral cord for the lower limb. It is not probable that these "segmental" nuclei contain "muscular" nuclei, *i. e.*, distinct cell groups for each muscle. The segmental division of the cells explains certain pathological processes: syringomyelia, progressive muscular atrophy, etc.

SPILLER.

Society Reports.

CHICAGO NEUROLOGICAL SOCIETY.

March 24, 1899.

The President, Dr. Richard Dewey, in the chair.

REMARKS ON CONTAGION AND INFECTION IN NERVOUS AND MENTAL DISEASES AND DEGENERACY, AND MEASURES OF PREVENTION.

The President read a paper with this title. He said that we do not ordinarily think of nervous and mental maladies as contagious or infectious, though they are literally so to some extent. It is, however, concerning *mental* contagion and infection that the writer wished to speak. Mental and nervous diseases may be, and often are, conveyed through the influence, direct or indirect, of mind upon mind. Nervous disorders like hysteria and chorea may spread from a single sufferer to an indefinite number of persons. The work of mental contagion is accomplished in a number of ways; it is immediate and direct, or slower in its working, involving a period of incubation and gradual evolution; or again, mental contagion and infection are spread by the process of procreation and inheritance. Examples of direct mental contagion are the epidemics so often recorded in history, especially in the middle ages, like those of flagellation, tarantism, the dancing manias and the convulsive epidemics, many of which phenomena continue in recent times.

Degeneracy may be considered one of the results of infection, and it is true that the human ovum or spermatozoid may be regarded as a microbe of all degenerative diseases, and this fact renders propagation of nervous and mental diseases by extension from generation to generation the most serious morbid influence with which we have to contend.

Mental contagion in its slower working is illustrated by the *folie à deux* of the French writers which indeed often includes three or four victims in one house or family. Dr. Dewey had seen five members of one family affected at the same time.

In all cases where nervous and mental infection or contagion occurs, we must recognize that the individuals are for the most part constitutionally predisposed.

In considering the question of prevention, it is often urged that the speedy removal of those capable of propagating nervous diseases and insanity would be justifiable. A more just view is, that although degeneracy, insanity and nervous diseases are injurious to-day, they are less mischievous than in former ages, and that the best remedy is not permanent seques-

tration, prohibition of marriage, castration and the like—measures which are fundamentally impracticable—but the constant effort of physicians and scientific men to enlighten the public mind.

A citizen of even moderate intelligence who would resist a compulsory law would, in many cases, avoid the penalties imposed by nature if brought to see the malign character of such penalties. It is probable that if the time ever arrives when legislation forbidding marriage, requiring castration or providing a peaceful death in incurables could be enacted and enforced, the time would also have arrived when it would not be needed. Public opinion will not tolerate legislation which risks the oppression of innocent persons, and although ten men might agree that the right of marriage should be denied in certain cases, or that certain individuals should be emasculated, yet, if one of the ten were in this way touched in his own person or family he would set the regulation at defiance, and the courts would probably sustain him in so doing. Human nature objects to being made virtuous "by acts of Congress."

It was, therefore, in Dr. Dewey's opinion, by education and instruction disseminated earnestly and incessantly throughout the community, that we have our chief means of combating these injurious agents, and not by attempting to regulate by statutes things which must be left to the growth of intelligence and mental and moral rectitude in the community.

Dr. Sanger Brown thought that although enlightenment and thorough education had gone far to eradicate the first noxious influence mentioned by Dr. Dewey, still the patronage at present bestowed upon Christian science and osteopathy showed that a considerable susceptibility yet remains even among individuals who have achieved more than the average of social and commercial success. Mankind *en masse* is prone to a peculiar paralysis of the reasoning faculties in certain directions which makes the individual commit himself to a course quite contrary to common sense. This is frequently seen in the enthusiasm aroused by an accomplished orator. An individual with sound and well-trained reasoning faculties can never be a genuine victim of this certain kind of infection, although he may at times simulate the condition.

Regarding the second division of the paper, he thought that as the environment of high civilization is constantly increasing in complexity, there will be more and more individuals who will fail to respond to it satisfactorily—more will fall by the way. It is true that educational, mechanical and business methods have been improved and simplified, but notwithstanding this, the tax levied upon individual energy has steadily increased. While some very high types are being developed, the process is horribly destructive and, he was sorry to say, that he could not share the sanguine hopes of the essayist that progress is being made toward a condition of more comfort and security.

Dr. H. N. Moyer was inclined to differ with Dr. Dewey regarding the increase of degeneracy, and thought that the apparent increase was due largely to the increasing interest in, and study of, the con-

dition; to the consequent increase of knowledge of the condition, and to the fact that civilization has become very complex, and that therefore, any aberration of mind is at once apparent and unfits the subject for the place which he should occupy. In a simpler condition of society, such an individual would pass entirely unnoticed, as do now many cases of mental disease in the rural districts. He believed that degeneracy and insanity had always been frequent, as they are still, even among the savage races and among the lower animals. The rogue elephant is a well-recognized instance of mental aberration, and such animals are always excluded from the tribe. Another element which must not be forgotten is the increased average longevity, so that many more people now reach the age when degeneracy of a certain kind manifests itself than was formerly the case.

Dr. Moyer was in hearty accordance with the essayist regarding the inefficiency of legislation for the cure of these evils, but thought that the tentative efforts which had recently been made in this direction showed an increasing recognition of many of the facts elucidated in the paper. When facts or theories have reached a point where they are accepted by more than a majority of a community, they will always find expression in legislation; but as legislation can never be in advance of public opinion, but must always follow it, and as these opinions are not yet widely disseminated in the community, nothing is to be looked for from legislation at present.

Another unrecognized tendency is towards socialism; individualism is becoming less and less a factor in legislation and in government, and the immense complexity of modern civilization is gradually forcing us into a modified state, socialism. It is increasingly recognized in certain countries; there is much socialistic legislation in Germany, not a little in Switzerland, and tentative efforts are apparent in the legislation of many of our Western States.

Dr. J. J. M. Angear was inclined to think that insanity frequently originated from mismatched marriages and family infelicities. Another fruitful cause, he thought, was excessive ambition, undue aspiration and excesses of all kinds that seem to be inseparable from our present civilization. These etiologic factors are not necessarily immediately causative, but having operated throughout several generations finally produce a neuropathic tendency, and ultimately the neuroses and psychoses.

Dr. Sidney Kuh called attention to the fact that rigid methods of limiting the spread of degeneracy would necessarily remove the milder types, and thought that their removal would entail a great loss to the public. He instanced the first four kings of Bavaria who were all degenerates, and yet did more for their country than the average sane crowned head of Europe. He reported several examples from his own experience, showing that some of the most brilliant achievements of the present day are attained by persons who are moderately degenerate, but who, nevertheless, make valuable contributions to literature, science and art.

Dr. Dewey in closing the discussion said that he had merely mentioned three ways in which contagion and infection are operative, and did not go into the question of their comparative importance in the development of degeneracy. On the whole, he seemed to consider heredity as the most important factor, and thought that degenerates were generally those who had been "infected" in the act of procreation, being imperfect by virtue of something derived from one or both parents. Incidentally he referred to the plan of "boarding out" the insane, and believed that the plan was vicious and should be prevented by law. A child brought up with an insane or idiotic person must necessarily be influenced by it.

Periscope.

PATHOLOGY.

202. ZUR KLINISCHEN PATHOLOGIE DES PERIPHEREN NERVENSYSTEMS BEI LUNGENTUBERCULOSE MIT SPECIELLER RÜCKSICHTSNAHME AUF AKROPARASTHESIEN (Contribution to the Clinical Pathology of the Peripheral Nervous System in Pulmonary Tuberculosis, with Special Regard to Akroparesthesia). Rudolf Schmidt (Wiener klin. Wochenschrift, Nos. 27, 28, and 29, pp. 721, 746, and 770, 1899).

Schmidt writes a lengthy paper on the alteration of the peripheral nerves in pulmonary tuberculosis, and quotes the opinions of many authors. His most important conclusions are:

1. The alterations of the peripheral nerves associated with pulmonary tuberculosis are:

(a) Disturbances of a local character, usually of a mechanical origin, more rarely of a toxic, dependent on the position of the nerve near the focus of infection (plexus brachialis, nervi intercostales, nervus recurrens, etc.).

(b) Disturbances from toxic substances producing more diffuse results.

2. Among the symptoms classed under (a) the phenomena of unilateral plexus pain on pressure and of homolateral akroparesthesia (often located in the distribution of the ulnar nerve) are worthy of special mention, as occurring especially in the early forms of pulmonary tuberculosis.

3. Tuberculosis, especially in youthful persons, must be remembered among the many causes of akroparesthesia.

4. The symptomatic akroparesthesia of phthisical patients is characterized by:

(a) Its frequency in males.

(b) Its frequent association with acute catarrhal pulmonary processes (influenza, bronchitis).

(c) Its unilaterality.

(d) The homolateral plexus pain on pressure.

(e) A parallelism with marked phthisical symptoms (night sweats, evening fever, etc.).

SPILLER.

203. DIE DEGENERATION DER MARKHALTIGEN NERVENFASERN DER WIRBELTHIERE UNTER HAUPTSÄCHLICHER BERÜCKSICHTIGUNG DES VERHALTENS DER PRIMITIV FIBRILLEN (Degeneration of Medullated Nerve Fibers). G. Mönckeberg and A. Bethe (Archiv. f. mikroskopische Anatomie u. Entwicklungsgeschichte, Vol. 54, 1899, p. 135).

The authors describe some results by new methods of investigation on the histological characters of the primitive fibrillæ in a number of animals, and the pathology of degeneration. They state that the axis cylinders of medullated nerve fibers in vertebrates consist of individual primitive fibrillæ, sharply outlined, and a homogeneous perifibrillar substance which surrounds them. Every fibril throughout the nerve fiber has a uniform caliber and has no knobs at the nodes of Ranvier. At such places the perifibrillary substance is absent, and thus has no place as a conducting element. The sheath of Schwann sends down processes at the nodes, and is broken by these as the myelin sheath is

divided. The first change in a nerve following a lesion is a change in the staining qualities of the fibrils; following this the fibrils themselves degenerate. The degenerated fibrils form large granular bodies, which break down into smaller ones, and later are absorbed. The perifibrillary substance shows granular changes. JELLIFFE.

204. WEITERER BEITRAG ZUR LEHRE VON DER ACUTEN NICHT-EITRIGEN ENCEPHALITIS UND DER POLIENKEPHALOMYEELITIS. (A Further Contribution to the Study of Acute Non-purulent Encephalitis and of Poliencephalomyelitis). H. Oppenheim (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, 1899, Nos. 1 and 2, p. 1).

An anemic girl, of sixteen years, had headache, loss of appetite, and gastric disturbance for several weeks. Headache became more severe, and vomiting, vertigo, chills and fever were added to the symptom-complex. The left abducens became paretic. The left side of the neck and head and the mastoid process of the left side were very sensitive to pressure, and the presence of empyema of the mastoid process and sinus thrombosis was suspected, although the otoscopic examination was negative. Operation revealed nothing abnormal in the region of the mastoid process, but four days later complete motor aphasia and right facio-brachial monoplegia developed. Other symptoms were noted. A diagnosis of non-purulent encephalitis was made, chiefly from the combination of focal signs with the symptoms of a general febrile disease. The patient died after a few months from purulent meningitis; and in addition to the lesions of this disease, two sclerotic foci—evidently the remains of lesions that had caused the focal symptoms—were found in the left third frontal gyrus and the foot of the anterior central convolution. This case shows that acute non-purulent encephalitis is not always fatal. The spontaneous pain and pain on pressure in the region of the left ear, that led to the mistaken diagnosis of mastoid disease, and the presence of abducens paresis, were interesting features in a case of encephalitis.

In a second case, in which the diagnosis of poliencephalomyelitis was made, the lesions were very slight. Diminution of the electrical reaction in the masseter muscles, incontinence of urine, absence of symptoms of exhaustion, and absence of remissions, were employed in diagnosing between poliencephalomyelitis and asthenic bulbar paralysis. A lymphosarcoma in the anterior mediastinum was believed to be the source of toxic products. SPILLER.

205. NOTES ON THE CHROMOPHILIC MATERIAL IN THE MOTOR CELLS OF BRAIN AND CORD, NORMAL (ANIMAL) AND PATHOLOGICAL (HUMAN), AND OF THE REACTION (ACID OR ALKALINE) OF THE CORTEX AND CEREBROSPINAL FLUID. John Turner (Brain, 22, 1899, p. 100).

The author presents the results of a number of studies on various points connected with the chemistry of chromophilic bodies. In a first series of observations he concludes:

1. Chromophilic material in the form of Nissl bodies is met with in animals immediately after killing and in human beings at least seventy minutes after death, when the nervous substance has merely been colored by a solution of methylene blue in normal salt without any further addition of reagents.

2. The fact that in some forms of diseased nerve cells the Nissl bodies show a gradual attenuation so that they dwindle down from bulky spindles and blocks to the finest imaginable threads is opposed to the idea that they are produced by precipitation after death.

3. The reaction of the cortex varies with different indicators, showing that it contains both acid and alkaline substances.

4. Its variations are constant, whether tested two hours or forty-four hours after death, and are as follows: acid to phenolphthalein and litmus, alkaline to methyl orange.

5. With animals killed and immediately tested, the gray matter gave similar reactions to phenolphthalein and methyl orange, but was alkaline to litmus.

6. The reactions of the cerebrospinal fluid differ from the cortex, in that both litmus and alkaline reaction is obtained. Otherwise they are the same. These results do not differ, whether the fluid is tested seventy minutes or forty-four hours after death.

7. Lactic acid is present always in cerebro-spinal fluid after death, and appears to increase with the length of the interval which has elapsed between the time of death and the time of testing.

In a second part he contributes some observations on mental cases of a cursory nature with conclusions which have been reached before by numerous other observers.

JELLIFFE.

206. THEORIE CEREBRALE DE LA RESPIRATION DE CHEYNE-STOKES (Theory of the Cerebral Origin of the Cheyne-Stokes Respiration). Rabé (La Presse médicale, May 13, 1890, No. 32, p. 227).

The first theory of this type of respiration was suggested by Traube, who believed that the respiratory center in the medulla was exhausted, and could only be stimulated by the accumulation of a large amount of carbon dioxide in the body. Filehne suggested that vasomotor innervation might have something to do with the condition. The apnea was the result of super-oxygenation, and the period of rapid respirations the result of an excess of carbon dioxide. Grasset suggested that the medullary centers were unduly susceptible to irritation, and reacted, therefore, with more than normal vigor; they became, however, readily exhausted, and the period of apnea corresponded to the stage of recovery. Franck called attention to the fact that in this type of respiration most of the patients were in a state of stupor, and Riche demonstrated that normally we inspire more air than is actually needed. Pachon subsequently undertook to study the respiratory movement among the insane, and found that the respiration rhythm was accentuated in the psychoses with hyperideation, or in cases of hallucinatory crises. In one patient, a woman suffering from melancholia and feeble intellect, the respiration was periodic. Pachon, therefore, suggested the cerebral origin of the Cheyne-Stokes rhythm, and called it a simple respiratory delirium. This theory accords well with the rhythm observed in various mental symptoms.

SAILER.

207. A STUDY OF LEUCOCYTOSIS ASSOCIATED WITH CONVULSIONS. F. G. Burrows (Am. Journ. Med. Sciences, vol. 117, 1899, p. 503).

The author from a few cases of convulsive seizures, in 3 cases of general paresis, 1 of catatonia, 1 of puerperal eclampsia, and three of various dementias, has drawn the conclusions that there is a leucocytosis associated with convulsive states not only in general paresis, but in various other diseases as well, and that the leucocytosis bears a relation to the severity of the seizure. He further claims that this leucocytosis is in part due to the muscular work done during a convulsion—a part which, owing to its physiological character and its short duration, has been shown to be superimposed upon pathological leucocytosis of longer duration.

JELLIFFE.

208. REMARKS ON THE PATHOLOGICAL CHANGES IN A CASE OF CHRONIC SYPHILITIC SPINAL PARALYSIS. R. T. Williamson (British Medical Journal, No. 1983, p. 1921, Dec. 31, 1898.)

A case which clinically corresponded to Erb's syphilitic spinal paralysis, and ran a gradually progressive course of five years, the first symptoms having appeared five years after infection.

In the pia mater and arachnoid evidences of slight meningitis were found in all regions of the cord, but the changes were most marked in the dorsal region, least marked in the cervical region. The meninges were slightly thickened by fibrous tissue, and in various parts there was slight infiltration with round cells chiefly around the blood vessels.

The spinal blood vessels presented well-marked changes in numerous parts. The larger arteries in the meninges, especially at the posterior part of the cord, near the posterior nerve roots, often presented marked endarteritis. The internal coat was often much thickened by abundant new cell formation on the inner side of the elastic lamina, the lumen of the vessel being thereby much diminished. The middle and external coats were generally not much altered; but frequently there was an infiltration of leucocytes around the vessels. The veins were either unaffected or the changes were less than in the arteries. In many cases the veins were surrounded by cell infiltration. The very small meningeal arteries presented thickening of the walls and slight proliferation of the cells of the intima, and they were often surrounded by round-celled infiltration.

Within the cord, especially in the diseased parts, the walls of the small vessels were often very much thickened, and had a homogeneous hyaline appearance, also the endothelial cells of the intima were slightly proliferated. In some places these hyaline vessels were surrounded by slight infiltration of leucocytes.

The two prominent features of the vascular changes were the endarteritis of the larger meningeal arteries and the hyaline thickening of the walls of the small vessels within the cord.

The cord showed ascending and descending degeneration with a gummatous infiltration of one lateral column in the dorsal region, several patches of intense degeneration and a band of degeneration at the periphery of the cord.

PATRICK.

CLINICAL NEUROLOGY.

209. POLYMYOSITIS. Sir W. R. Gowers, (British Medical Journal, No. 1985, p. 65, Jan. 14, 1899).

The affection consists in a simultaneous inflammation of many muscles and of some nerves, and is closely allied to polyneuritis. It is met with almost exclusively as a result of exposure to cold. It seems to be a peculiar variety of the rheumatic poison produced in specially susceptible individuals, rendered such by some influence which depresses the general health. In such cases of multiple inflammation of both the muscles and the nerves, the affection presents the general distribution of polyneuritis so far as preponderant loss of power is concerned, but with a far wider implication of the muscles, of which many suffer that escape in ordinary polyneuritis. They are at first very tender, and afterwards undergo hardening and contraction, which may be extreme in degree; and indeed, after a time, may be insuperable, and resist all efforts to overcome it.

The patient whose case is reported was a woman thirty-six years of age, with a rheumatic heredity. The first symptoms were a con-

stant tired feeling and aching pain in the back and around the loins. An irritable rash appeared over the hands and arms. Then she began to have pain with weakness in the hands and ankles, both pain and weakness increasing in degree and extent until they involved nearly all the voluntary muscles in the body and the patient was absolutely helpless. The muscles became rigid, and she had to be moved in bed as one stiff piece. The extremities were fixed in flexion with wrist-drop and foot-drop. All the muscles reacted slightly to strong electric stimulation, faradic or galvanic. Sensation was normal. No deep reflexes could be obtained. Perspiration was excessive. The affection ran a slowly progressive course for about two years when some slight improvement began and, at the time of the report, about two years and a half after the beginning of the trouble, she was able to walk a little, but the upper extremities were still practically useless. The author thought that she would ultimately be able to walk reasonably well. He thought that the time for treatment is during the acute stage or soon after the onset, when rest, diaphoretics and salicylates, with perhaps small doses of mercury, would control the inflammation and lessen very much the subsequent mischief.

PATRICK.

210. LA MALADIE DE BLAISE PASCAL (Blaise Pascal's Disease). Binet Sangle (Annales medico-psychologique, 1899, March).

This psychological study of Pascal here brings out a number of interesting facts. The family history, as to his ancestors, is lacking; yet it is certain that his brothers and sisters were decidedly neurotic and hysterical and were all short-lived.

It seems evident that Pascal suffered from severe neurasthenia, in that he complained frequently of transitory paraplegias, general prostration, persistent digestive disturbances and obstinate headache. He was extremely emotional, at times hypochondriacal, and had a number of phobias with hallucinations. Moreover, he had distinct periods of disordered judgment, which coincided with grave alterations in his general health.

BENOIT.

211. A CASE OF GENERAL PARALYSIS OF THE INSANE IN A CHILD. John Thomson and D. A. Welsh (British Medical Journal, No. 1996, p. 784, April 1, 1899).

The patient was a girl and there was distinct evidence of hereditary syphilis. When she was between ten and a half and eleven years of age it was observed that she was not improving in her school work, and seemed to be getting stupid and irritable at home. No further change was noted in her mental condition until she was about twelve, when fits set in, and she became steadily less intelligent. Her speech was characteristically affected by the time she was twelve and a half years of age, and her knee-jerks were greatly exaggerated when she was thirteen years of age. When about fourteen years of age she had several distinct hallucinations. Between eleven and fourteen years of age she became unnaturally fat, but afterwards she steadily emaciated. The fits continued at varying intervals during her whole life. Six months before her death she had an attack of subacute periostitis over the right tibia, which was greatly relieved by iodide of potash. She died in a state of extreme debility of mind and body, aged sixteen years and eleven months.

The post-mortem examination showed no trace of subdural membrane, but an opaque and milky pia-arachnoid everywhere adherent to the brain and an excess of cerebro-spinal fluid. The cerebral con-

volutions were greatly atrophied, being small, narrow, and separated by dilated sulci. Atrophic changes were general, but most marked in frontal and parietal regions of each side. On section the cortical gray matter was found to be extremely atrophied, its layers indistinct, and its consistence tougher than normal. The white matter showed irregular patches of congestion and increased toughness. The basal ganglia were also congested. The lateral ventricles were somewhat dilated, and their ependyma showed fine scattered granulations; more numerous and more prominent granulations were present in the fourth ventricle. The choroid plexus showed no obvious change. Microscopic sections of the cortex from various regions showed changes characteristic of general paralysis in the nerve cells, in the neuroglia, and in the vessels. PATRICK.

212. SUR UN CAS DE COXALGIE HYSTERIQUE (A Case of Hysterical Hip-Joint Disease). M. Lannois (Lyon médical, Vol. 90, p. 395).

The patient was a young peasant woman, of twenty years, who was subject to violent hysterical convulsions. Although the family and personal history, as well as the previous course of the affection, indicated tubercular hip-joint disease, the author found hyperesthesia, or rather hyperalgesia, in the region of the hip, affecting skin and muscles, and these structures were much more sensitive than was the joint to heavy percussion over the trochanter. Under chloroform anesthesia the joint structures were found to be entirely normal. A novel method of cure was the plan pursued of demonstrating the free mobility of the joint to a number of co-patients, who afterwards ridiculed to the subject the idea of her being really disabled. This ridicule brought on a violent hysterical attack, which was followed by complete disappearance of the previous symptoms. PATRICK.

213. TETANOID SEIZURES IN EPILEPSY. L. Pierce Clark (American Journal of Insanity, 55, 1899, p. 583).

Tonic or tetanoid epilepsy, Clark holds, judging from literature, and his experience at Craig Colony, to be a comparatively rare condition, though he is not disposed to make a special type of epilepsy of such tetanoid seizures. The history of a case is presented, who prior to the time of the observations had had grand mal seizures. In the tetanoid seizures the head was first slightly rotated to the right, and then decidedly to the left. Then the spasm passed rapidly over the entire body, apparently bilaterally from head to foot in a wave like manner. All the muscles were in tonic rigidity each time for fifteen to twenty seconds. The back was well arched as in tetanus; the muscular rigidity being so great that the body could be raised from the floor, by lifting the ankles, the occiput supporting the upper end. These tonic spasms occurred for a period of three hours, and were not intermingled with those of a clonic nature. JELLIFFE.

214. UN CAS D' EPILEPSIE JACKSONNIENNE HYSTERIQUE (A Case of Hysterical Jacksonian Epilepsy). J. Crocq (Journal de Neurologie, August 20, 1899, p. 321).

Attacks of convulsions developed in a man after a fright, and became as numerous as twenty-five in twenty-four hours. They began with tingling in the left leg. Tonic spasm of short duration in this limb gave place to clonic spasms, and these involved successively the left arm and head and later the right side, but predominated still in the left side. The face, mouth and eyes were drawn to the left, con-

sciousness was not lost, and one sentence was repeated several times during the attack. The attack lasted a half minute to a minute and a half. No foam was seen at the mouth, and no incontinence of urine was detected. Some hyperesthesia of the left side was observed. In addition to the seizures just described, the patient had attacks of tingling beginning in the left leg or arm and becoming general. He suffered also from vertigo and headache located on the right side. Percussion of the cranium revealed a painful area over the right Rolandic region.

A lesion of the right Rolandic area, especially in the center for the lower limb, was suspected. It was believed to be of a tuberculous nature, on account of the history of tuberculosis in the family. Operation seemed to be demanded, but suggestion was first employed. Crocq discoursed to his students in the presence of the patient upon the employment of nitrate of silver in epilepsy, and gave directions for its administration with much attention to detail. The suggestion was successful. The attacks began to diminish in frequency at once, and the headache and painful area soon disappeared. The patient soon left the hospital, and after some weeks wrote that he was cured. The case was believed to be one of hysterical Jacksonian epilepsy. SPILLER.

215. UN CASO DE PAQUIMENINGITIS CERVICAL HIPERTROFICA (A Case of Hypertrophic Cervical Pachymeningitis). Mariano Alurralde (Anales del Circulo Médico Argentino, No. 4, Feb., 1899, p. 107).

Under this title Alurralde reports a case of a carpenter, thirty-six years of age. The family and previous history were unimportant. At the onset of the disease ten months previously the patient suffered from pain in the ring and little finger of the left hand lasting three days. The pain then diminished in intensity here, and involved successively the wrist, elbow, shoulder and scapula. Four months later the neck was attacked, and a small but rapidly growing tumor, painful on pressure and of hard consistency, was noted in the median and left lateral portion of the neck. Its appearance was soon followed by intense cephalalgia, pronounced weakness, drooping of left lid, deviation of the mouth, and slight weakness of the left lower extremity. Fowler's solution was given for forty days without appreciable effect. The tumor, which was thought to be a sarcoma of the ganglia, was then removed. The wound healed in nine days, and in three months the cheek, eyelid, left arm and left leg had recovered to such an extent that the patient was soon able to return to work, at which he remained for four months. At the end of this time pain (in the neck) became so severe that he was compelled to abandon work and re-enter the hospital. On entrance his general condition was poor, the head was held rigidly bent forward, as well while he was at rest as when in motion, and it was found that this position was due to contractures of the neck muscles. The spine was tender to pressure as far down as the mid-dorsal region, and the pain was increased by movement. The left upper extremity showed, besides the typical *main en griffe*, with its accompanying atrophy of the thenar, hypothenar and interosseal muscles, diminution in the volume of the muscles of the forearm. In the left shoulder-girdle group there was atrophy of the deltoid, pectoralis major, the supra and infra-spinatus and the inter-scapular muscles, the latter causing separation of the left scapula. R. D. was present in the muscles most affected, and quantitative changes both to faradism and galvanism in the others. The deep and superficial reflexes were normal, and sensation was not affected. The left pupil showed slight myosis and both reacted sluggishly in accommodation and to light, the defect being most marked with regard to the latter. The temperature

on the left side was 33° C., on the other 34° C. This latter side showed no apparent diminution in muscular volume, but the dynamometer revealed considerable weakness.

The patient soon began to have difficulty in deglutition and phonation, with some pain in the pharynx, and a retropharyngeal abscess was found and evacuated. Eight days later the same procedure again became necessary. In his differential diagnosis, the author excludes torticollis, rheumatism, progressive muscular atrophy and syringomyelia, and believes the condition to be one of hypertrophic pachymeningitis, due to suboccipital spinal caries. The question of a metastatic process following the primary growth in the neck is not even alluded to.

J. W. COURTNEY.

216. QUELQUES CONTRIBUTIONS A L'ÉTUDE DU SOMMEIL CHEZ LES SAINS D'ESPRIT ET CHEZ LES ALIÉNÉS (Sleep in the Sane and in the Insane). Pilcz (Annales medico-psychologique. Jan., 1899, p. 1).

During the past five years the author has been observing this condition, and concludes that the soundness of sleep for many of the insane is similar to that found in normal people, and that this soundness of sleep is closely correlated with dream states. The more profound the sleep, the less liability to dreams; in less profound sleep, the dreams take on the character of old associations and old ideas, whereas in light sleep, new impressions gain entrance into the mental processes and influence the character of the dream. The author holds that the delusions of the insane are rarely reproduced in their dreams.

JELLIFFE.

217. UEBER HEREDITÄREN TREMOR (On Hereditary Tremor). Graupner (Deutsches Arch. f. klin. Med.; 64, 1899. Festschrift).

The history of a neuropathic family is here given. Three of six sisters were affected in middle life with a marked intention tremor; in one of these choreic movements of the facial musculature were also manifest. A daughter of one of these patients was affected from early childhood by a tremor of the upper extremities, and was subject to much muscular weakness on very slight exertion. The author suggests the relationship of the cases described by him to Huntington's chorea.

JELLIFFE.

218. A CASE OF BRAIN TUMOR AT THE BASE OF THE SECOND LEFT FRONTAL CONVOLUTION. H. C. Gordinier (Am. Journ. Med. Sciences, 117, 1899, p. 526).

Gordinier here presents a case of unusual interest, which seems to show that there exists at the base of the second left frontal convolution, for right-handed persons, a cortical center for writing analogous to the cortical center for motor speech. A like center for the other side probably exists. Destruction of this center produces pure motor aphasia without aphasia, and with no paralysis of the arm. The case should be consulted in the original.

JELLIFFE.

THERAPY.

219. ON THE PHYSIOLOGICAL ACTION OF CHOLINE AND NEURINE. F. W. Mott and W. D. Halliburton (British Medical Journal, 1899, March).

The cerebro-spinal fluid removed from cases of brain atrophy, particularly from cases of general paralysis of the insane, produces when injected into the circulation of anesthetized animals a fall of

arterial blood pressure, with little or no effect on respiration. This pathological fluid is richer in proteid matter than the normal fluid, and among the proteids, nucleo-proteid is present. The fall of blood pressure is, however, due not to proteid, nor to inorganic constituents, but to an organic substance which is soluble in alcohol. This substance is precipitable by phospho-tungstic acid, and by chemical methods was identified as choline.

The nucleo-proteid and choline doubtless originate from the disintegration of the brain tissue, and their presence indicates that possibly some of the symptoms of general paralysis may be due to auto-intoxication; these substances pass into the blood, for the cerebro-spinal fluid functions as the lymph of the central nervous system. The authors have identified choline in the blood removed by venesection from these patients during the convulsive seizures which form a prominent symptom in the disease.

Normal cerebro-spinal fluid does not contain nucleo-proteid or choline; or, if these substances are present, their amount is so small that they cannot be identified. Normal cerebro-spinal fluid produces no effect on arterial pressure; neither does the alcoholic extract of normal blood or of ordinary dropsical effusions.

The presence of choline in the pathological fluids will not explain the symptoms of general paralysis; for instance, it will not account for the fits just referred to. Its presence, however, is an indication that an acute disintegration of the cerebral tissues has occurred. If other poisonous substances are also present, they have still to be discovered.

Neurine, an alkaloid closely related to choline, is not present in the fluid; its toxic action is much more powerful, and its effects differ considerably from those of choline.

PHYSIOLOGICAL ACTION OF CHOLINE.

The fall of blood pressure caused by choline is in some measure due to its action on the heart, but is mainly produced by dilatation of the peripheral vessels, especially in the intestinal area. The action on the splanchnic vessels is due to the direct action of the base on the neuromuscular mechanism of the blood vessels themselves; for after the influence of the central nervous system has been removed by section of the spinal cord, or of the splanchnic nerves, choline still causes the typical fall of blood pressure. The action of peripheral ganglia was in other experiments excluded by previous intravenous injection of a solution of nicotine. Section of the vagi produces no effect on the results of injecting choline. The effect of choline soon passes off, and the blood pressure returns to its previous level.

PHYSIOLOGICAL ACTION OF NEURINE.

The doses employed varied from 1 c.cm. of 0.1 per cent. solution. These were injected intravenously.

Neurine produces a fall of arterial pressure, followed by a marked rise, and a subsequent fall to the normal level. Sometimes, especially with small doses, the preliminary fall may be absent. Sometimes, especially with large doses, by which presumably the heart is more profoundly affected, the rise is absent. The effect of neurine on the heart of both frog and mammal is much more marked than is the case with choline; in the case of both choline and neurine, the action on the frog's heart is antagonized by atropine.

The slowing and weakening of the heart appear to account for the preliminary fall of blood pressure; in some cases this is apparently combined with a direct dilating influence on the peripheral vessels. The rise of blood pressure which occurs after the fall is due to the constriction of the peripheral vessels, evidence of which was obtained by the use of oncometers for intestine, spleen, and kidney. After the in-

fluence of the central nervous system has been removed by section of the spinal cord, or of the splanchnic nerves, neurine still produces its typical effects. After, however, the action of peripheral ganglia has been cut off by the use of nicotine, neurine produces only a fall of blood pressure. It therefore appears that the constriction of the vessels is due to the action of the drug on the ganglia; in this, it would agree with nicotine, coniine, and piperidine. Section of the vagi produces no influence on the results of injecting neurine. In animals anesthetized with morphine and atropine, injection of neurine causes only a rise of blood pressure, which is accompanied with constriction of peripheral vessels.

Neurine produces no direct results, so far as could be ascertained, on the cerebral blood vessels. Neurine is intensely toxic to nerve trunks (Dr. Waller and Miss Sowton). It produces a marked effect on the respiration. This is first greatly increased; but with each successive dose the effect is less, and ultimately the respiration becomes weaker and ceases altogether. The animal can still be kept alive by artificial respiration.

The exacerbation of respiratory movements will not account for the rise of arterial pressure; the two events are usually not synchronous, and an intense rise of arterial pressure—due, as previously stated, to contraction of peripheral blood vessels—may occur when there is little or no increase of respiratory activity, or during artificial respiration.

As confirmatory of Cervello's statement that neurine acts like curare on the nerve endings of voluntary muscle, and to which he attributes the cessation of respiration, it may be mentioned that after an animal has been poisoned with neurine, asphyxiation causes little or none of the usual convulsions.

It should be mentioned that in the case of brain atrophy referred to the cerebro-spinal fluid was removed soon after death. Since then the authors have had the opportunity of examining two specimens removed during life by lumbar puncture, and the results corroborate the conclusions previously arrived at.

PATRICK.

220. ON THE TREATMENT OF ABDOMINAL PALPITATIONS. Sir Willoughby Wade (British Medical Journal, No. 2007; June 17th, 1899, p. 1451).

This well known disorder, most common in women, but not infrequent in men, is in all cases unpleasant, often distressing, and in some instances painful, so much so that patients are most grateful for relief. Palpation of the abdomen during an attack, and often in the intervals, reveals an abnormally forcible pulsation of the abdominal aorta. From theoretical and practical considerations, it seemed to the author that the cause was to be found in a state of high arterial tension, and he considered that nitroglycerine would be the logical remedy. He says that his surmise proved to be correct on trial, but he has apparently tried it in but few cases. He administers one two-hundredth of a grain every night and finds that although the supposedly increased tension in the abdominal aorta is diminished, the pulse at the wrist is at once rendered more full and forcible.

Several cases are adduced in support of the treatment, but they appear to the reviewer to fall somewhat short of constituting invulnerable evidence.

PATRICK.

Book Reviews.

DIE PSYCHIATRISCHE KLINIK ZU GIESSEN. Ein Beitrag zur praktischen Psychiatrie von Dr. A. Dannemann, Oberarzt der Klinik. Mit einem Vorwort von Prof. Dr. Sommer, Director der Klinik, mit zwölf Grundrissen. Verlag von S. Karger, Berlin, 1899, pp. 120.

The reviewer has read the book and also visited this new psychopathic hospital during this summer's vacation; so that he may speak with some enthusiasm of the institution and the brochure which describes it. All the German university towns now have a psychopathic hospital, a psychiatric clinic, with the single exception of Kiel. The plans are already made for one at Kiel, however, and a year or two from now will see a clinic of the same order there, modeled upon the one at Halle. This at Giessen is as yet the newest in Germany. It was completed and opened for patients in 1896. There is room for about 100 patients in eight villas, four for each sex. The patients are separated into private, quiet, restless and disturbed classes, and a villa provided for each class. In addition to these buildings for patients, there is a central kitchen building, a cottage for the director, and the central or administration building, making in all eleven separate structures. These are symmetrically arranged in pleasant gardens without fences. The central building is perhaps most interesting to the neurologist and alienist, since it is here that Prof. Sommer carries out such investigations as he describes in the volume recently issued by him, entitled "Lehrbuch der Psychopathologischen Untersuchungs Methoden." In this building are the administration offices, clinical auditorium, out-door department or dispensary, autopsy room, pathological laboratory, chemical laboratory, psychophysical laboratory, photographic room, library, workshop for the manufacture of mechanical and scientific apparatus, and rooms for the assistant physicians. Giessen is a town of only fifteen or sixteen thousand inhabitants, and the value of land is not great, so that the psychopathic hospital was enabled to expand over a considerable area. When we come to build such clinics, as we needs must do ere long, or be left out of the current of progress, we shall have less space, especially in our large cities, but the same end can be attained by compressing our psychopathic institute into one building. I have no doubt that we shall surpass Germany when we once begin this reform, at least in structural arrangements, if not in devotion to the scientific side of morbid psychology. The scientific part will develop later, when we have the stimulus of chairs of psychiatry in our medical colleges and of psychiatric clinics and laboratories. Those who are interested in insanity, in the best methods of care of the insane, and in the means and methods of investigation, should read this book, and have it at hand when they are called upon to suggest plans for the psychopathic hospitals which are about to be erected in many of our university towns.

FREDERICK PETERSON.

A TEXT BOOK OF MENTAL DISEASES, WITH SPECIAL REFERENCE TO THE PATHOLOGICAL ASPECTS OF INSANITY. By W. Bevan Lewis, F.R.C.P. (Lond.), M.R.C.S. (Eng.), Medical Director West Riding Asylum, Wakefield; Lecturer in Mental Diseases at the Yorkshire College; Examiner in Mental Diseases to the Victoria University. Second Edition. P. Blakiston & Co., Philadelphia, 1899.

Bevan Lewis' "Mental Diseases" has in a peculiarly attractive sense occupied a foremost rank in the literature of psychiatry. For the Eng-

lish reading student it has been one of the most comprehensive of treatises, and in its pathological features it may be claimed with justice that it contained the most exhaustive and complete summary that could be obtained, apart from serial publication literature or special monographic treatments. Bearing in mind the manifest difficulties in book making, from a publishers' standpoint, and when second editions are involved, it may be stated that the present volume is a fairly complete working over of the first edition, now some ten years old, though the book is not by any means a new one, nor has the recasting been done in a systematic way, but obtains more thoroughly in some places than in others.

The general method of arrangement remains the same. The book is divided into three sections: Part I, Anatomical and Histological Section. Part II, Clinical Section. Part III, Pathological Section. In part I, the histology of the cerebral cortex has been more thoroughly treated than in the first edition, and for the size of the volume, a fairly complete summary of present day knowledge of the structure of the ganglion cells is given. The illustrations are augmented by a few Golgi preparations, but the author has not given any place for the more recent histological details of the ganglion cells proper. The summary seems to bear the stamp of literary culling rather than of actual work done with the newer histo-chemical methods; the aniline blue black method still remains the *pièce de résistance*. The only good cytological illustration is from Lenhossek, of the spinal ganglion cell, so widely copied by all writers.

Section II on the clinical features remains much the same as in the first edition. It is one of the best and most complete, possessing clearness and terseness in description, if lacking the philosophic charm of Maudsley. There is such a broad ground for logical differences in point of view regarding the classification of mental diseases, that little need be said in praise or condemnation of any particular method of grouping the symptom complexes, yet in the author's treatment of the various types of delusional insanities, monomanias, paranoias, and allied psychoses under the general term of "Mental Enfeeblement" we lack the usual perspicacity of a good general grouping. The subject matter of the clinical section has not been materially altered, and we miss, what to us are cardinal points in the development of new conceptions regarding the psychological aspects of mental states, any reference to the works of hosts of contemporary alienists, notably Kraepelin, Ziehen, Mendel and Krafft-Ebing.

In the section on pathology the evident lack of attention to the work of others produces a marked blemish in a book of this character. The illustration of the animal "that went into his hole and then drew it in after him," is not altogether inappropriate for the insularity displayed by the author in his treatment of the pathology of the nervous system. We have carefully gone over the whole ground and fail to find any recognition, save for a few foot-note references, of later day performances of real merit bearing on the general subject; and we cannot feel that justice has been done to the subject or to the reader in offering a section so markedly one-sided, so lacking in the recognition of much knowledge done by scores, even hundreds of other observers, and so bound down with the weight of the importance of a dominant idea, the "scavenger cell." For a summary of our present day knowledge of the pathology of the nervous system in insanity the present section is far from satisfactory. Whether author or publisher is responsible for this show of stagnation of ideas we do not presume to decide, but certainly it would seem to

show, in science, an insularity, if not aloofness from contemporary thought, so frequently commented on of Englishmen in other spheres of work.

The mechanical get-up of the book is all that could be desired, and were the work permeated with conceptions of a later date that have been tried and found true, it would remain the classic that it has deservedly been.

JELLIFFE.

NERVOUS AND MENTAL DISEASES. By Archibald Church, M.D., Professor of Clinical Neurology and of Mental Diseases and Medical Jurisprudence in the Northwestern University Medical School (Chicago Medical College); Professor of Neurology in the Chicago Polyclinic; Neurologist to St. Luke's Hospital, Chicago, etc.; and Frederick Peterson, M.D., Clinical Professor of Mental Diseases in the Woman's Medical College, New York; Chief of Clinic, Nervous Department, College of Physicians and Surgeons, New York. With 305 Illustrations. W. B. Saunders, Philadelphia, 1899.

There is no doubt that in the present instance the authors modest claim of a carefully prepared text-book is fully borne out. Text-books conform in their general scope to the multifarious types of those who may use them. Some are written down to the low level of quiz compendianism, others shoot far above the average student's knowledge, if not his actual brain capacity to utilize the facts; a third class of book, to which this conforms in high degree, is one that gives the student a carefully arranged series of facts, sufficiently extensive and aptly descriptive to conform to most types of disease with which he, as a student, and later, as a general practitioner, is liable to meet and be called upon to diagnose and treat in this special line. There are some very commendable features in these treatises, for each part of the work is sponsored by the respective authors, rather than its being a conglomerate. In the opening chapters of Dr. Church's part on neurology, the chapters on methods of examination are very full, practical and well illustrated. The usually extensive and often cumbersome chapters on the anatomy of the nervous system are, and wisely, we believe, omitted, and a feature of prominence is the transferral of such anatomical matter to its discussion in résumé under each disease. Thus, the anatomy of the fifth nerve is briefly taken up under diseases of that nerve; the general morphology of the brain, with cerebral localization and brain lesions. This serves to give the reader a clearer picture than if he should have to look up the anatomy of a region from insufficient data in opening chapters of a text-book, or from Gray or Quain or Morris, with its elaborate fulness.

The descriptions of the various diseases are unusually terse and characteristic.

Dr. Peterson's part of the work upon mental diseases embraces in part matter already published by this writer in medical periodicals. The author's chapter upon the so-called stigmata of degeneration is excellent, both in regard to subject matter and illustration; it probably forms the best text-book exposition of this subject of our day for the busy practitioner.

The classification and description of the different varieties of insanity are very well and clearly given. Notwithstanding the present prevalent belief that the best possible clinical pictures of the insanities

have been published, we see much that is to be highly commended in the author's presentation of epileptic insanity, and especially in the forceful delineation of paranoia.

The chapter on idiocy and imbecility is undoubtedly the best that has been given us in any work of recent date upon mental diseases. The photographic illustrations of this part of Dr. Peterson's work leave nothing to be desired.

JELLIFFE.

THE BULLETIN OF THE OHIO HOSPITAL FOR EPILEPTICS. Vol. I, Nos. 2 and 3. July and December, 1898. Published by the Hospital, Gallopis, Ohio.

The first number of the *Bulletin* from the Ohio Hospital for Epileptics was evidence of good work done in that institution, and the volume before us now confirms the favorable impressions created by the first appearance of the periodical. Dr. Rutter, the manager of the hospital, writes a short and interesting analysis of the statistics of the hospital since its opening, in 1893, to the present time, but the book is essentially the work of the pathologist, Dr. Ohlmacher. Anyone familiar with the details of pathological study will recognize at a glance that these papers are the result of much careful work, of thought, of long hours spent with the microscope, and of extensive reading. While, therefore, we may not all be prepared to accept Dr. Ohlmacher's views, we may read his statements with respectful attention, and with the willingness to be convinced.

Dr. Ohlmacher has been in charge of the pathological laboratory of the hospital for a little over two years, and during this time necropsies have been held on twenty-five epileptic patients. A valuable pathological material has thus been collected. The lymphatic constitution, from his studies, acquires an importance in epilepsy that it has not previously received from the pen of any other writer. When his first report on this subject was published, not quite two years ago, he was unable to find any reference to similar observations. Dr. Ohlmacher approaches this subject in the spirit of a scientist. He says: "I am willing to have all these observations received with scientific conservatism, or even skepticism, if need be."

Let us look at what Dr. Ohlmacher has really demonstrated. He has reported five additional cases (three previously published) of genuine grand mal with prominent evidences of the lymphatic constitution. These were the most typical examples of "idiopathic" epilepsy in nineteen epileptics examined after death. The remaining fourteen cases included examples of secondary epilepsy, epileptiform convulsions of idiocy, infantile paralysis, etc., not to be included with primary grand mal; and of genuine epileptic insanity with death after gradual wasting, where, presumably, the once present adenopathies of the lymphatic dyscrasia had atrophied and disappeared. The most prominent anomaly in these five cases was a persistent thymus, with other evidences of the lymphatic constitution; in three of these cases signs of rachitis, and in four of thyroid disease, were present.

Dr. Ohlmacher has attempted to convince the most skeptical that the morbid anatomy of the lymphatic constitution is associated with idiopathic grand mal in the majority, if not all, cases which exhibit the typical characteristics of this form of epilepsy, but he does not attempt to explain to us the nature and origin of this lymphatic constitution and the manner in which it acts. He admits that in his former paper the liberty of speculation with only three cases as support for his conclusions may have been abused; now that he has the pathological findings in eight cases, his opinions have more basis. He desires to show a morphological

and clinical resemblance between genuine epilepsy, rachitis, eclampsia infantilis, thymic asthma and thymus sudden death, tetany, and possibly exophthalmic goiter; and the common basis of these affections is the lymphatic constitution. We do not find any distinct statements in regard to the cases of petit mal. Are these also dependent on the lymphatic constitution? The inference would be that they are, as in many instances petit mal later develops into grand mal. According to Ohlmacher, the anomalies of the lymphatic constitution may disappear in some cases of idiopathic epilepsy, either by the growth changes of maturity, or by the wasting from prolonged debility.

It is important to have clearly in mind the essential features of the lymphatic constitution; these are, the persistent and enlarged thymus, the general lymphadenoid hyperplasia, the arterial hypoplasia, evidences of old rachitis, the involvement possibly of the bone marrow, and possibly the alteration of the thyroid.

This seems to be a clear and correct presentation of Ohlmacher's views. Has he shown any relation of cause and effect between the lymphatic constitution and idiopathic epilepsy? It seems merely that in eight cases which he regarded as the most typical of idiopathic epilepsy this constitution was present. Back of this constitution there may be still another cause which produces both the lymphatic constitution and the manifestations of idiopathic epilepsy. This possibility was evidently present in Dr. Ohlmacher's mind, as he employs the words "associated with" in speaking of the constitution and the epilepsy. His main attempt has been to emphasize the association of the morbid anatomy of the "status lymphaticus" and epilepsy.

Ohlmacher's studies will awaken interest in the subject he has so ably presented, and we shall not wait long for confirmation or refutation of his views. Epilepsy, unfortunately, is widespread, and the necropsies are not very rare. We shall doubtless have some further observations from pathologists on the lymphatic constitution in epilepsy, and the findings in a larger number than eight cases will permit more certainty in forming conclusions.

In another paper we have the description of two cases of cerebral tumor. Ohlmacher enters into a discussion concerning the relation of the lymphatic constitution to cerebral glioma. His words are so striking that they are quoted: "* * * * this would mean that gliosis and glioma are the neuroglial tissue changes induced by a lymphatic poisoning (in which the thymus perhaps plays the dominant rôle); that the gliosis of epilepsy, and in consequence epilepsy itself, are to be referred back to a similar autotoxic cause." This is interesting speculation, but so far as we know it it not fact, and Ohlmacher does not present it as such. Are we quite prepared to accept this hypothesis? We need more than the occurrence of the lymphatic constitution in a "goodly percentage of cases of idiopathic epilepsy where gliosis in one form or another is also present."

In one of the cases of cerebral glioma ptosis of the left eyelid was found. This condition has been seen before in cases of cerebral tumor, and Ohlmacher's case, like the others, does not satisfactorily explain the phenomenon. In the same paper the close resemblance sarcoma may bear to glioma is clearly illustrated.

A few shorter papers are found in this number of the *Bulletin*, together with the description of an interesting case which has been described as a "pathological museum," and which is familiar to the readers of the *JOURNAL OF NERVOUS AND MENTAL DISEASE*.

SPILLER.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

RIGIDITY OF THE SPINAL COLUMN.*

BY PHILIP ZENNER, A.M., M.D.,
CINCINNATI.

In 1893 Bechterew¹ reported three cases of rigidity and curvature of the spine with peculiar nervous manifestations, considering them to present a special form of disease. Whilst no attention had hitherto been paid to this subject in neurological literature, a number of other publications was called forth by that of Bechterew, the cases being alike, at least, in the presence of rigidity of the spine, though how far they all belong in the same category of disease is doubtful. As the matter has not, so far as I know, received any consideration in American neurological literature, I wish to give a brief review of published cases, as well as to report a few coming under my own observation.

Bechterew² in a second article with the report of additional cases, gives the following résumé of the symptoms presented:

Complete, or nearly complete immobility of a part of, or the entire spin.

A large rounded posterior curvature of the spine, chiefly in the upper dorsal region. The head appeared to be pushed forward or sunken while the part of the spine below the pos-

* Read by title at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

¹ "Steifigkeit der Wirbelsäule und ihre Verkrümmung als besondere Erkrankungsform." *Neurol. Centbl.*, 1893, p. 426.

² "Von der Verwachsung oder Steifigkeit der Wirbelsäule." *Deutsche Zeitsch. f. Nervenheilkunde* Bd., xi., p. 327.

terior curvature was straight, the normal concavity in the lumbar region being absent.

A series of symptoms that may be looked upon as spinal root-symptoms, present to a varying extent in different cases, such as paresthesia and pains in the extremities, neck, trunk and spine; in a few cases, cramp-like contraction especially in the extremities; impaired sensation in areas supplied by the lower cervical, dorsal, or lumbar nerves; weakness of the muscles of the neck, trunk and extremities; and usually, slight atrophy of scapular muscles; usually little or no pain produced by either percussion, or attempted movement, of the spine.

The electrical reaction of the spinal muscles was sometimes lessened quantitatively, but not otherwise affected. There was some flattening of the chest, and, mostly, abdominal breathing. The joints of the extremities were only occasionally affected. The disease was in all instances slowly progressive, and, while offering no hope for improvement, did not appear to have any direct fatal tendency. His patients were all fifty years old or more, excepting one who was about thirty-five. Heredity or trauma appeared to be the cause in some instances.

Bechterew, while knowing of no autopsies, expressed the opinion that there was progressive ankylosis of the spine, involvement of the spinal roots, and extension of the inflammatory process to the outer layer of the dura and the surrounding connective tissue.

The series of cases reported since Bechterew's have mostly another very pronounced condition, ankylosis of the hips. In some there was disease also of other joints. In his second article Bechterew stated that the only other report similar to his own with which he was acquainted was that in Oppenheim's text-book, under the heading arthritis deformans of the spine. According to the latter the disease is attended by pain in the spine, intensified by effort to move, and spinal root-symptoms attributed to slow compression through growth of bone into the vertebral canal such as intercostal, brachial, and crural pains, and atrophic paralyses. The disease runs a chronic course, with remissions and exacerbations. The diagnosis is based on arthritis in the joints, rigidity of a large part of, or the entire spine, which does not disappear under chloroform, and the spinal root-symptoms.

In the same journal an article of Strümpell's³ appeared in which he gives a clinical picture based upon three cases seen by himself. He describes the disease as remarkable and peculiar, resulting in gradual ankylosis of the spine and hips, without spinal root-symptoms, in fact with almost entire absence of subjective symptoms. One case was examined under chloroform. The spine remained immobile. Rotation of the hip, which was only slightly movable, produced crepitation. The spine was abnormally straight, instead of having the large kyphotic curve of Bechterew's cases. The muscles along the spine appeared abnormally hard and atrophied. His patients were between thirty and forty years of age. He says he knows of no autopsy in such cases, but, on account of the crepitation of the hip in one of his cases, believes that there is a chronic inflammatory process affecting the joints of the vertebræ as well as the hip, yet is in doubt whether this is in etiological relation with other arthritic processes.

Baumler⁴ in a later issue of the same journal gives a brief account of a case seen by him in 1873, with his opinion as to the nature of the disease. A man of twenty-three had inflammation of both hip-joints at seventeen; the lower part of the spine becoming affected, perhaps, at a later period. When seen by Baumler, in addition to rigidity of the spine and hips and lessened mobility of the right shoulder, the head was bent forward, there was thickening of the tissues about the cervical spine, and the patient suffered with constant pain in the neck.

Baumler believes that the state of the spine in these cases is due to mechanical causes. He says, when the spine of normal individuals is held long in one fixed position, on account of the occupation or the like, local pain and tenderness are produced which require a long rest for their removal. He thinks that in the kind of cases we are considering, inflammation of the hips or of part of the spine, and, possibly in addition, a strained position due to the special occupation, cause the as yet unaffected spine to be held in a fixed position, with no normal wholesome

³ "Bemerkung über die chronische ankylosirende Entzündung der Wirbelsäule und der Hüftgelenke." *Deutsche Zeitsch. f. Nervenheilkunde* Bd., xi., p. 338.

⁴ "Ueber chronische ankylosirende Entzündung der Wirbelsäule." *Deutsche Zeitsch. f. Nervenheilkunde* Bd., xii., p. 177.

play of the parts. As a result there are firstly, abnormal circulatory conditions, congestion in the immobile parts, and, subsequently, from the presence of micro-organisms, inflammation. In his own case the patient was a writer, and the rigidity of hips and larger part of spine required a constant strained position of the cervical spine—a forcible bending over in order to read his books—and inflammation about the cervical spine ensued. After a long rest the inflammation and pain about the neck disappeared, and the man resumed his work, though the rigidity remained. Baumler supposes that such mechanical influences were the chief features in Bechterew's cases, whilst the same, with an additional inflammatory concomitant, would explain his own and Strümpell's cases. In others, he thinks the condition may be due to inflammation alone.

Marie⁵ reports two cases, and gives as characteristics of the disease, complete rigidity of the spine, with more or less pronounced ankylosis of the hips and shoulders (therefore "rhizomélique"), the small joints remaining free. In his cases the hips were quite ankylosed, there was lessened mobility in shoulders and knees, a pronounced rounded posterior curvature of the upper half of the spine, the lower half straight without the normal concavity in the lumbar region; there were exostoses on the sacrum and cervical spine, flattening of the chest and atrophy of muscles in the dorsal and gluteal regions.

Mutterer⁶ reports a case like Marie's in the condition of the spine, hips and shoulders. The soft parts around the cervical spine were decidedly hard, long muscles of the back flaccid and atrophied, and gluteal muscles atrophied. The knees were swollen, there was no tenderness over the vertebrae, and nothing abnormal, exostoses, etc., to be detected. The patient, a man of fifty-eight, was often kept awake at night by shooting pains in his legs. Otherwise he was in good health. His first symptoms were pains in the loins eight years before. Four years later there was some rigidity, shooting pains in the legs and some difficulty of walking. The rigidity slowly passed from below upwards, the cervical region being last affected, its involvement being accompanied by severe pain.

⁵ "Sur le spondylose rhizomélique." *Rev. de Médecine*, April 10, 1898.

⁶ "Zeitschr. f. Nervenheilkunde Bd., xiv., p. 144.

Popoff⁷ reports a case in a man of twenty-three, distinguished from the other reported cases in its rapid development. The disease was apparently due to the patient's being in the water five hours fishing. The same evening he had fever and pain in the loins and knees. Within two months, during which time there was pain in the spine, there developed the condition of the spine noted in Bechterew's cases, rigidity, large kyphotic curve in its upper half and absence of normal convexity in the lumbar region. There was tenderness over the spine, from the sixth dorsal to the third lumbar vertebra. He continued to have occasional pains in the knees, sides and spine. The breathing was abdominal, the gait weak and tremulous. Though the spine was rigid the head could be moved freely.

This case is unlike the others, excepting Bechterew's, in the freedom of the joints. But during his illness the thumb was swollen and painful for a period of six weeks, and, six years prior to this period, the patient had had inflammation of the wrists for a year, terminating in permanent ankylosis, all pointing to arthritic tendencies.

In how far all of these cases belong to the same category of disease is by no means clear. Bechterew and most of the other authors speak of the absence of post-mortem findings, but Bechterew⁸ has since reported the death from pneumonia of one of his cases, and the post-mortem examination. The very brief report of this examination, under society proceedings, merely states that there was very slight mobility of the spine, that the bodies of the upper dorsal vertebræ were grown together to a larger or smaller extent, that there were osteophytes on the bodies of these vertebræ, and that there were no marked changes in the dura. The posterior roots of some of the dorsal nerves were degenerated. There was a slight grayish discoloration of the root zones of the posterior columns.

We may gain some light on the subject from surgical literature.

Marsh⁹ mentions a case of Fagge in Guy's Hospital in

⁷ "Ueber die Ankylose der Wirbelsäule." *Neurol. Centbl.*, 1899, p. 294.

⁸ *Neurol. Centbl.*, Feb. 1, 1899, p. 143.

⁹ "Rare Forms of Bony Ankylosis," *Brit. Med. Journal*, June, 1895, Vol. 2, p. 1087.

1874—a man thirty-four years of age. A year prior to this time the spine became stiff and formed a rounded curve. The right hip became ankylosed subsequently. The patient died of chest trouble.

At the autopsy there was found ankylosis of the right hip, of the attachments of the ribs to the vertebræ, as well as complete ankylosis of the arches, spinous and articular processes of the dorsal vertebræ. The vertebræ were softened so that they could be cut with a knife. The spine was fragile.

Marsh states that bony ankylosis of the spine is common in osteo-arthritis. Such specimens are to be found in every large pathological museum.

The union results from the direct fusion of component bones, as in Fagge's case, from ossification of the ligaments, or from the formation of bony buttresses connecting the adjoining vertebræ, the joints remaining unaffected. Marsh states, also, that all large museums show specimens of bony ankylosis in extreme lateral curvature, the vertebræ being fused together in the concavity of the curve.

Koehler¹⁰ in an article on an unusual case of spondylitis deformans, describes a case in many respects like the foregoing. A man of sixty-one, six years prior to Koehler's seeing him, noticed some stiffness in the legs, and, soon afterwards, in the spine. The mobility of the knees and hips became gradually lessened, their movement being attended by a creaking sound, and a feeling as if sand were in them. Movement of the head was attended by a sense of cracking in the upper cervical vertebræ. There was some pain on attempted movement, otherwise scarcely any pain. At the end of four years, spine, hips and knees were nearly rigid. At this time he fell on the floor in walking and broke his left thigh in its upper third. He had been in bed six months when first seen by Koehler. At this time he was very emaciated, especially in the muscles of the back, which required a very strong faradic or galvanic current to produce contractions. There was no tenderness over the spine, slight lateral curvature in the dorsal region, but no deformity. The head could be turned or bent to the least possible extent. Otherwise the entire spine from

¹⁰ *Charité Annalen*, 1887, p. 619.

head to sacrum, was found, under chloroform, to be as rigid as a board. On examining by way of the throat there was found to be a number of irregular swellings of the size of beans on the sides of the cervical vertebræ. Osteophytes were also felt upon the sacro-iliac joints. The left hip was totally ankylosed. The right hip and the knees were very slightly mobile, their movement producing crepitation.

Koehler gives the following description of spondylitis deformans. The intervertebral disks fibrillate or are entirely destroyed; sometimes there is distinct growth of new cartilage. Furthermore, supra-cartilaginous exostoses grow on the rims of the vertebral bodies, in part in stalactitic form. They may become united with like growths from the neighboring vertebra, forming a bony clasp which connects the vertebræ together. Rarely is the entire rim of the vertebra involved in this process. Usually only the lateral aspect, or only one side is affected. At the same time there is atrophy of the body of the vertebra, different parts being affected to different degrees, so that it assumes an irregular form as regards height, breadth and depth, and as a result there may be strikingly irregular curvature of the spine. At the same time the ligaments may undergo a bony transformation. This deforming inflammatory process may attack single vertebræ, a number of vertebræ, or even the entire spine.

Koehler quotes Braun as giving the causes of spondylitis deformans in fifty-eight cases—in most of which there was disease of only a few vertebræ—as follows: Syphilis 3, trauma 2, arthritis deformans 3, gout 4, in the rest chronic rheumatism. Koehler believes in his own case the presence of exostoses, and appearance of the joints prove that the disease was arthritis deformans, notwithstanding that this disease almost never leads to ankylosis. The ankylosis of the left hip is due, in his opinion to the long immobility following fracture of the thigh.

Before commenting further on the disease, I will add the report of some cases of my own.

Case 1. M. R., age twenty-nine, merchant. His father, who suffered with diabetes, died of obstruction of the bowel at fifty-eight. His brother and himself are color-blind. No his-

tory of rheumatism in the family. No history of trauma. Excessive venery, no tobacco, nor alcohol.

Nine years ago he had gonorrhea. There was a suspicion at the time of the presence of a urethral chancre, but, so far as he knows he has never had any constitutional symptoms of syphilis. He gives a history of varied pains for a long period of time, even antedating the just mentioned venereal disease. He does not give a very good account or description of these pains, but he has had at times pains in the back, in the neck, the extremities, and, probably is never for a long period altogether free from pain. That in the back seems to have been mostly of the character of lumbago. The pain of which he complains most is of a severe tearing character in the neighborhood of the right loin, coming on in paroxysms, and occurring chiefly at night. Such paroxysms of pain were what especially led him to consult me. He thinks he has observed some stiffness of the back for five years, and that it has tended to increase. He has also had various choreiform symptoms; for eight or ten years, occasional "tic" of eyelids; for a year, occasional "tic" of neck, and also peculiar noises coming from the throat, which are at times made unconsciously, again as a result of a certain sense of constraint. Otherwise he has always enjoyed excellent health.

The examination of the patient apart from the "tics" just spoken of, reveals nothing abnormal, excepting in what relates to the spine. The latter appears to be quite immobile, excepting the upper cervical vertebræ. The head can be rotated freely, and can be bent backwards and forwards, though not quite as much as normally. There is a rounded kyphotic curve in the upper dorsal region, but probably not so large as that depicted by Bechterew. The normal concavity in the lumbar region is absent. When he stands the shoulders are thrown a little backwards, the abdomen and knees a little forwards, the knees slightly flexed. The whole attitude is that of a peculiar bent-over posture. The stiffness of the trunk and all, also gives the gait a peculiar appearance. The muscles of the back respond normally to the electric current, and they do not appear abnormally hard or atrophied. There is no tenderness over the spine. The patient can jump on his heels without producing any pain. He is accustomed to walk long distances with ease. He presents a few symptoms of a spastic character. At times he cannot cross the knees with ease. He cannot fully extend the knee while in a sitting position, apparently on account of strong contractions of the hamstring muscles. Occasionally he feels a sudden contraction of the muscles of the trunk so that for a moment he cannot straighten the body as

well as usual. He always has observed that he is taller in the morning, on rising, than later in the day.

The patient, who came from a southern city, remained under my charge for several months. The treatment consisted mainly in maintaining the supine position for several hours each day, hot applications to the spine during that time, and massage of the muscles. He suffered more or less with pains, mostly of the character of muscular rheumatism, but not severe. He had no recurrence of the paroxysms of severe pains from which he had been suffering. His general health remained good. When he left, the rigidity of the spine, and its appearance remained unchanged.

Case 2. M. S., age forty-four, policeman. A sister has epilepsy. Much addicted to tobacco, not to alcohol. Denies syphilis, but states that he had iritis in 1892. Has been married fifteen years and has healthy children.

In 1884, when his duties were very taxing, and he was subjected to much exposure, he was laid up with lumbago for six weeks. In 1894 "caught cold" and was again laid up with pains in his back and a stiff back. He has never been altogether well since the latter date. His chief complaint has been of pain. This pain is mostly in the loins, chiefly on the left side, and is only felt when making a movement, especially if trying to arise while on his back. He says he awakes with pain several times in the night, possibly the result of turning in his sleep.

As in the first case, apart from what relates to the spine there are no objective symptoms, the muscular strength, cutaneous and muscular sensation, reflexes, special senses, etc., being normal. He presents the same condition of the spine: immobility of all parts excepting the upper cervical region, and a kyphotic curvature in the upper dorsal, and lower cervical region. The head appears pushed forward, though not drooping. The movement of the head is much less than in my first case, there being scarcely any lateral movement, and little flexion or rotation. The kyphotic curve is not marked and the normal lumbar lordosis not altogether lost. The man was originally six feet two and one-half inches in height, now he measures one and three-fourth inches less. There is some tenderness over the vertebræ, especially the lumbar, but greater tenderness over the muscles of the loins. The breathing is distinctly abdominal. Though a tall man the measurements of the chest show only one inch difference between forced inspiration and forced expiration.

This patient was also under observation for several months, with no perceptible change excepting that the tenderness over the spine and muscles disappeared.

Case 3. The third case appears to be altogether different from the other two. The patient is a boy sixteen years of age, brought to me to examine into his mental condition. He had spasms in infancy, and has always been somewhat peculiar in disposition. It is needless for me to enter here into a consideration of his mental condition. Suffice it to say that while he is honest and conscientious, learns easily, has a good memory, etc., he has many mental stigmata of degeneration; is abnormally sensitive, stubborn, given to violent fits of anger, slovenly to the point of indecency, etc. He has also many physical stigmata, thick lips, prominent ears, strabismus, very awkward hands, chest pigeon-breasted, etc. He is also a somnambulist.

He has a large kyphotic curve of the spine, taking in the whole dorsal region. There is, at the same time, no mobility, or very little of the entire spine, excepting in the upper cervical and lumbar regions. On account of the curvature the head is pushed forward, but it is freely movable. There is the normal lumbar lordosis. The breathing is free and normal. There is no tenderness over the spine or back, and he does not suffer, as far as I can learn, has never suffered, with pain in these parts. I could not obtain any satisfactory history of how long this curvature had existed.

The feature common to all the cases mentioned was rigidity of the spine, either a large part of, or the entire vertebral column being involved. In most instances the ankylosis affected also the costo-vertebral joints, indicated by the abdominal breathing. The large kyphotic curve was also a common feature, though absent in Strümpell's, Baumler's, and Koehler's cases. Koehler believed that it always appeared after some time, and was only absent in his own case on account of the patient's long confinement to bed, an explanation which is scarcely satisfactory.

Spinal root-symptoms, spoken of by Oppenheim and Bechterew, were not noted or not pronounced in most instances, and, in general, pain was not very marked.

That the disease was of a distinct arthritic character in some instances seems clearly indicated by the condition of other joints—complete or partial ankylosis, sometimes crepitation—and, in a few cases, by the presence of osteophytes on the vertebræ. It may be that they differed in the character of the arthritis; gouty, rheumatic, arthritis deformans, etc. That all the cases were primarily arthritic is not so clear, not of Bech-

terew's, Popoff's, and my own. Popoff mentioned a case of his own, reported in an earlier publication, in which the curvature of the spine was decidedly reduced under chloroform. The curvature was also lessened by lying a few hours, and it was noticed that the spine was straighter in the morning than in the evening, yet no mobility of the spine could be detected on examining him. In this instance Popoff believed the spinal rigidity was from muscular contractions. In his other case there had been some lessening of the curvature of the spine during the four months the patient was under observation. In this case, in which the whole process was of rapid development, the curvature began to appear within a month of the onset of pain and gradually increased. But it was observed that in the beginning the curvature would disappear when the patient lay upon his back, and only after some months, became permanent.

In my first two cases it impressed me as if the trouble were primarily muscular. The pains were chiefly of the character of muscular rheumatism, there were no joint symptoms, and the first patient spoke of always being taller in the morning when he got up than in the evening, and presented other spastic symptoms.

Mutterer¹¹ quotes Beer, whose report of a like case was inaccessible to me, as saying that the soft parts were the chief seat of the disease.

If muscular contractions be the primary condition in these instances it is not improbable that bony ankylosis finally ensues either like that occurring in lateral curvature, or from a resulting inflammation in the vertebræ or intervertebral disks.

¹¹ Zeitschr. f. Nervenheilkunde, Bd., xiv., p. 144.

ALVEOLAR SARCOMA OF THE RIGHT MIDDLE FOSSA OF THE SKULL.*

BY MORRIS J. LEWIS, M.D.

F. H., female, æt. 11 years, was brought to the dispensary of the Orthopedic Hospital and Infirmary for Nervous Diseases by her mother, for right facial palsy, on January 11th, 1899, and gave the following history:

Father died at the age of 35 of left hemiplegia.

Mother living and in good health at the age of 36. Three brothers living and well.

Previous History:—Ordinary diseases of childhood. Diphtheria in 1896.

Present History:—In the latter part of August, 1898, patient had a right lower molar tooth extracted for decay, considerable pain followed this, and very shortly afterward she began to have right-sided earache. There was no discharge from the ear at any time, but in three weeks after the extraction of the tooth, paralysis of the right side of the face gradually made its appearance, and steadily increased until it became total.

About December 4, 1898, symptoms of irritation of the right eye made their appearance, and subsequently a small ulcer developed on the cornea.

When the child applied for treatment the face was completely paralyzed upon the right side, and strongly drawn to the left, the child being utterly unable to close the right eye, which was very much injected and prominent, probably from relaxation of the eye muscles. The cornea was the seat of a small ulcer.

The tongue was thrust to the left, the uvula projected to the right, and the child was unable to chew upon the right side of the mouth. The sense of taste was less acute upon the right side of the tongue than upon the left, particularly on the anterior and middle third. The left tonsil was considerably enlarged.

There was, and had been for some time, pain in the right mastoid and temporo-parietal regions, particularly towards night.

The child was perfectly bright and answered all questions intelligently, and showed no signs of paralysis other than those mentioned.

*Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

Electrical examination made at the time of application, by Dr. Rhein, revealed as follows:

Right side of face,	6 ma.	A.C.C. > C.C.C.
Left side of face,	2 ma.	C.C.C. > A.C.C.

No transmission down the nerve to galvanism or faradism, and no response whatever on the right side of the face to faradism.

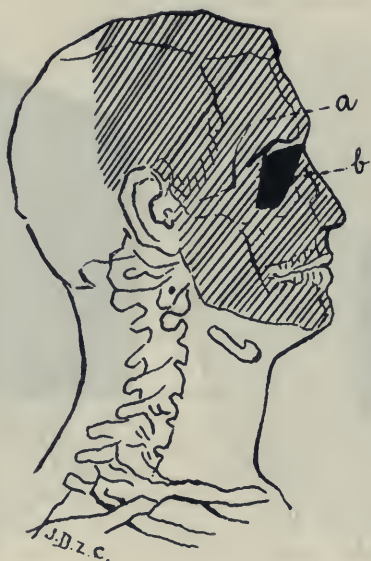


Fig. I. The diagram shows impaired sensibility extending over right side of face, with complete anesthesia above and below eye, and including the eye. *a* = impaired sensibility; *b* = total anesthesia.

Examination of the ears by Dr. Randall revealed: "Evidences of past suppuration, a depressed adherent scar of the right membrana tympani, with preponderant bone conduction, which is slightly exaggerated. But little congestion and no evidences of carious troubles, past or present."

The child was placed upon iodide and bichloride of mercury, with bromide of sodium at night for sleeplessness.

January 27, 1899. Dr. A. Thomson reported the following condition of the eyes:

"The right eye little better than it has been. Ulcer healing a trifle more, however. The cornea is completely vascular, with large shallow ulcer with infiltration of lower layers. Com-

plete anesthesia of cornea and of every part supplied by the fifth nerve. There is also marked involvement of muscles supplied by the third nerve, however, not complete. Has been using mydriatic, so dilatation of pupil cannot be relied upon. Internus affected most.

"O. S. pupil reacts normally, media clear. The disc swelled about 1 D., the margin cloudy and veins tortuous. Marked evidences of starting neuritis. No hemorrhages. No hemianopsia. Muscles all right in this eye."

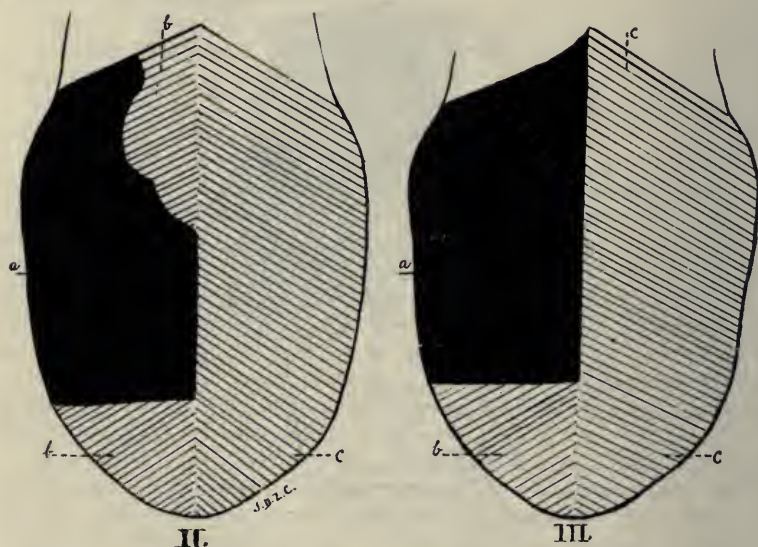


Fig. II. The diagram shows the condition of sensation in the tongue, Jan. 28, 1899. *a* = complete anesthesia; *bb* = impaired sensibility; *c* = normal sensibility.

Fig. III. The diagram shows the condition of taste in the tongue, Jan. 28, 1899. *a* = absence of taste; *b* = impaired taste; *cc* = normal taste.

January 27, 1899. Admitted to hospital, where the following notes were taken: Absolutely no evidences of paralysis, either of motion or of sensation in the extremities or trunk. Grip good, the right hand showing 25 and the left 22 on the dynamometer. Knee-jerks and elbow-jerks normal. Impaired sensibility of the whole right side of the face and of scalp to vertex, with complete anesthesia above and below the eye, and including the latter. Excessively tender spot in the deep layer of the upper lip upon the right side. Teeth of the upper and lower jaws do not meet evenly in the middle line, the

lower jaw being thrust to one side. The tongue is protruded towards the paralyzed side, being thrust over in its entirety. Slight edema of the scalp and forehead exists. The right eye is much worse, right internal rectus decidedly weak.

She was ordered iodide of potash in increasing doses, with inunctions of mercurial ointment. The eye was washed frequently with boric acid.

January 28th, 1899. Examination of the tongue showed complete anesthesia of the right side, except the tip and a small area towards the root, where the sensation was impaired, while in regard to taste there was complete absence on the right side except a small portion at the tip where it was impaired. (See diagram.) Marked tenderness exists to-day over the whole mastoid area.

February 1, 1899.—Patient much brighter. A second examination by Dr. Thompson reveals: "O. D. better. Cornea very much vascularized, but not so much discharge. O. S. pupil reacting normally (no Wernicke's sign). The disc seems a little more swollen, about 3 D. at summit now. Has had some subjective sensations on right side of face."

February 2, 1899.—This afternoon, after having been apparently as well as usual and conversing rationally with her mother, she gave a shriek, put her hand to her head over the right occipital region, and complained of great pain. No convulsion occurred, but for the most of the afternoon she uttered a typical meningitic shriek, and had some retraction of the head, and talked irrationally. The next morning she awoke fairly comfortable with no retraction of the head or headache, and talked rationally, the events of the night before being a total blank. In the evening the symptoms of the night before were repeated.

For the next ten days the patient had alternate periods of rest and pain, but her vision gradually grew worse, and she had great difficulty in distinguishing objects or telling the time by the watch. Her speech also became drawling.

From the 12th to the 17th she was bright again, and had no pain. Her speech became less drawling than before, but this drawling appears to be partly natural.

On the 17th she had intense burning pain on the right side of the face, although the paralysis did not appear to be as marked as before. This last symptom was noticed even by the brother.

On the 20th the hearing was much worse, and there was marked paralysis of the right external rectus muscle. Slight nystagmus was noticed for the first time. Hard tapping over

the right temporal region caused no pain whatever, although a short time later this procedure did cause pain.

On the 24th Dr. Thomson reports: "Choked disc of the left



IV.—Photograph of the brain with the tumor in position.

eye very greatly increased and pupil greatly contracted." She died in the evening of that day.

This case is particularly interesting on account of the pe-

cular grouping of the symptoms. It will be seen by studying the report of the case that the second, third, fifth, sixth, seventh, eighth and ninth nerves were all more or less involved upon the right side, while there was absolutely no discoverable involve-

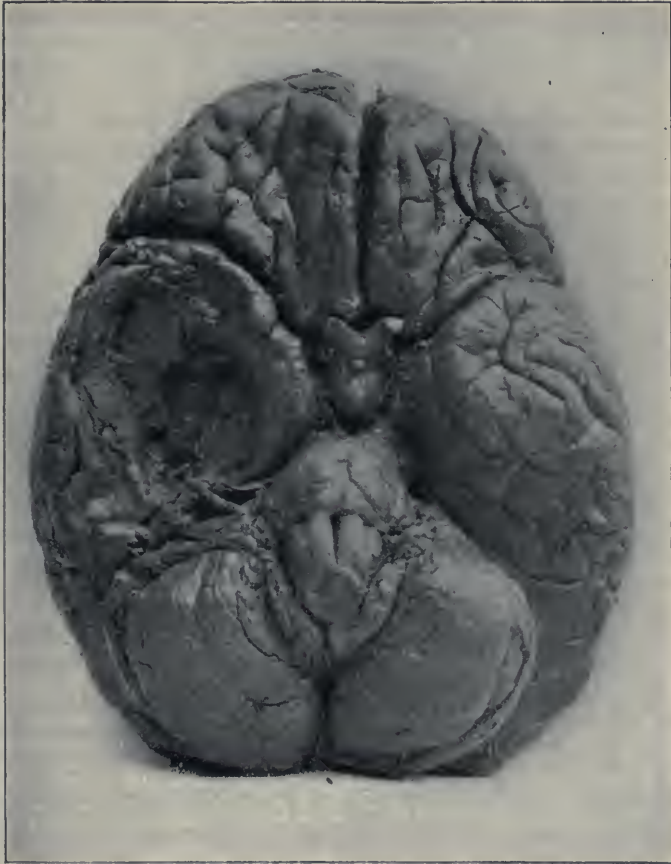


Fig. V.—Photograph of the brain after the tumor had been removed.

ment of any other portion of the nervous system. The tentative diagnosis of tumor of the right middle fossa of the skull was made and the autopsy proved this to be correct. My col-

league, Dr. Chas. W. Burr, made the autopsy, the report of which is as follows:

POST-MORTEM EXAMINATION BY DR. CHAS. W. BURR.

The necropsy was made some hours after death. The brain alone was examined. The scalp and calvarium were normal. On opening the skull the dura bulged a great deal. The longitudinal sinus was empty. The pia was dry and the convolutions on both sides much flattened and pale. The right middle fossa was filled by an irregularly globular mass, or rather by two fused into one, the larger being below the plane of the dura, the smaller above. The mass was two and a quarter inches in height, two and a half inches in width, and two and a half inches in length in its largest diameters. The upper surface of the mass had caused a depression in the basal surface of the right temporo-sphenoidal lobe, about one inch deep and one and a half inches wide. This surface was smooth, covered with pia, and had pushed the brain substance upward in front of it. The mass was shelled out from the brain easily. The under, larger part of the mass had eroded, and, indeed, invaded, the floor of the middle fossa, was firmly attached to it, and appeared in the roof of the pharynx on the right side. The dura surrounded the circumference of the mass between the larger and the smaller parts, and penetrated it a short distance. On section the mass was hard, firm and smooth. There were two areas within it about half an inch square, which were translucent and glassy in appearance. Microscopic examination proved the mass to be an alveolar sarcoma, probably arising in the dura. There was some, but not very much, hydrops of the ventricles.

221. DEMONSTRATION OF PRIMITIVE FIBRILLÆ. A. Bethe. (Arch. f. mikroskopische Anatomie, 54, 1899, p. 141).

In an exhaustive article on the characters of the primitive fibrillæ in nerve fibers, Bethe gives a series of micro-technical methods for staining the same. He employs osmic acid as a fixative, using small strands of nerve tissue, for about twenty-four hours. These are then washed 4-6 hours and then placed in 90 per cent. alcohol ten hours. Water four hours, and then transfer to a 2 per cent. solution of sodium bisulphate to every ten drops, of which 3-4 drops of concentrated hydrochloric acid has been added, for 6-12 hours. Specimens then can be washed, run up in alcohol and oil to paraffine, embedded and cut very thin. Fastened to slide by an albumin fixative and stained as follows: One-tenth of 1 per cent. warm, 50-60 C., toluiden blue for ten minutes, wash, and fix for a minute in a 1 per cent. solution of ammonium molybdate. Mount in balsam.

JELLIFFE.

A CASE OF INTERNAL HEMORRHAGIC PACHYMEINGITIS IN A CHILD OF NINE YEARS, WITH CHANGES IN THE NERVE CELLS.¹

By WILLIAM G. SPILLER, M.D.,

PROFESSOR OF DISEASES OF THE NERVOUS SYSTEM IN THE PHILADELPHIA POLYCLINIC; ASSOCIATE IN THE PEPPER LABORATORY; PATHOLOGIST TO THE PENNA. TRAINING SCHOOL FOR FEEBLE-MINDED CHILDREN.

AND

D. J. MCCARTHY, M.D.,

ASSOCIATE IN THE WILLIAM PEPPER LABORATORY OF CLINICAL MEDICINE; ASSISTANT IN NEUROLOGY IN THE PHILADELPHIA POLYCLINIC.

The existence of internal hemorrhagic pachymeningitis in childhood has received comparatively little attention in the text-books and general medical literature. Herter's² excellent article published in 1898, with the report of two cases, is an exception to the rule. Even when mention is made of the subject it is not usually in the report of cases or of personal experience, but merely in allusion to the writings of others.

Internal hemorrhagic pachymeningitis, even in the adult, is said by Gowers to be "very rare," and is of interest "chiefly as a pathological curiosity enigma." Gowers³ further states that its rarity outside of asylums may be judged from the fact that during the first forty years in which the London Pathological Society received the curiosities of metropolitan necropsy not a single specimen was brought before the society from any London hospital.

Osler⁴ says that during eight years in the post-mortem room of the General Hospital of Montreal no case of internal hemorrhagic pachymeningitis occurred, and the first specimen he saw after having been for fourteen years interested in mor-

¹ From the William Pepper Laboratory of Clinical Medicine, University of Pennsylvania (Phœbe A. Hearst Foundation).

Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

² Herter, *The American Journal of the Medical Sciences*, 1898.

³ Gowers, "A Manual of Diseases of the Nervous System." Second English edition, p. 325.

⁴ Osler, *Journal of Nervous and Mental Disease*, 1888, p. 608.

bid anatomy was in Virchow's laboratory; but he saw numerous examples later at the Philadelphia Hospital, usually in bodies from the insane department.

While the condition is not one of great frequency, it does occur often enough to be seen by many neuropathologists, and the mere report of a case in the adult would hardly be justifiable. The occurrence of this pathological condition in children is worthy of mention. According to Gowers, in early life it is relatively as well as absolutely infrequent, and occurs chiefly in infancy, as frequently in the first year of life as between one and twenty years of age.

Dercum,⁵ in an article in his text-book, refers to the papers by Doehle and Northrup as evidence of the occurrence of internal hemorrhagic pachymeningitis in childhood. He speaks also of a variety of chronic external pachymeningitis occurring in childhood, and described by Lannelongue and Alexis Thompson, and he mentions inherited syphilis as a cause of pachymeningitis in childhood.

Mills⁶ refers to the existence of the condition in children, the subjects of hemorrhagic rachitis, and in the discussion of a paper by Osler on internal hemorrhagic pachymeningitis reported a case in a child of about three years of age.

Northrup⁷ has reported four cases occurring in children, the eldest child being four years and seven months old. He refers to a case in childhood recorded by Emmett Holt, and to one by S. J. McNutt. He says he has seen only four cases, all in children under two years of age, in an experience founded upon fifteen hundred necropsies.

Hoyt⁸ says that the few cases reported as occurring in early life were due to traumatism or some grave general disease.

Bullard⁹ speaks of *traumatic* cases occurring in well-grown children.

Holt¹⁰ says that chronic internal pachymeningitis is not

⁵ Dercum, "A Textbook on Nervous Diseases," pp. 350, 352.

⁶ Mills, "The Nervous System and its Diseases," p. 265. *Journal of Nervous and Mental Disease*, 1888, p. 611.

⁷ Northrup, *Proceedings of the N. Y. Path. Soc.*, 1890, pp. 59 and 67.

⁸ Hoyt, *Medical Record*, April 30, 1892, p. 485.

⁹ Bullard, *Boston Med. and Surg. Journal*, Nov. 7, 1895, p. 461.

¹⁰ Holt, "Diseases of Infancy and Childhood."

very rare, being usually discovered at autopsy in children, chiefly cachectic infants, who have died of other diseases. He says he has seen five cases. He also makes the statement that pachymeningitis plays a much more important rôle in the production of meningeal hemorrhage in children than has generally been accorded to it.

Oppenheim¹¹ describes a form of chronic meningitis in young children, confined almost entirely to the posterior cranial fossa and supposed to be of syphilitic origin. The meninges are united with the cerebellum and medulla oblongata. This condition mentioned by Oppenheim seems to be different from that observed by us in the case to be reported in this paper.

According to Henoch,¹² pachymeningitis in moderate intensity and extent occurs not so very rarely in children, but is not as common as in adults. Our case seems to be extraordinary, not only on account of the advanced age of the child, but also on account of the extent of the process.

The oldest child with internal hemorrhagic pachymeningitis to which Herter was able to refer was six years of age (Doehle). The age of the patient studied by us was nine years.

Doehle¹³ believes, from the necropsies made in the pathological institute of Kiel, that chronic internal pachymeningitis is not so rare in children as is usually believed. In 395 autopsies on children, 269 being in children one year old and under, chronic pachymeningitis was found 48 times; and in 38 of these cases the children were not over one year old; in the other ten they were not over eight years old. No pachymeningitis was observed in children nine or ten years of age, and no mention is made of ages more advanced than these. Doehle even believes that pachymeningitis is of more common occurrence in childhood than in later life, but he states that the existence of chronic pachymeningitis in childhood has received little attention.

This brief résumé of the literature presents a variety of

¹¹ Oppenheim, "Lehrbuch der Nervenkrankheiten," first edition, p. 490.

¹² Henoch, "Vorlesungen über Kinderkrankheiten," 1892, p. 285.

¹³ Doehle, "Verhandlungen des X internationalen medicinischen Congresses," Abtheilung XVII, Gerichtliche Medicin, p. 40.

opinions held in regard to the occurrence of internal hemorrhagic pachymeningitis in childhood. No mention is made of the condition in certain books devoted to the diseases of children.

The case that we report is from the Pennsylvania Training School for Feeble-Minded Children. We are indebted to Dr. M. W. Barr and Dr. Llewellyn for the clinical notes.

M. F., born December 12, 1889, was the child of healthy parents. The father was a teacher and very erratic. The parents, though not related before marriage, bore a striking resemblance to one another. The father was thirty-six, the mother thirty-three, when M. was born. The parents and grandparents were not addicted to the use of alcoholic drinks. A history of insanity or feeble-mindedness was obtained in the family of a great-grandfather. There was no tendency to blindness or deafness in the child's family on either side, excepting that the father was somewhat deaf in the left ear as the result of a severe attack of scarlet fever when seventeen years old. Two sisters of M. were said to be of sound health in body and mind. She had no brothers. A dead sister, three years M.'s senior, resembled M. in constitution; and died in a home for feeble-minded children in Iowa, of "tuberculous meningitis," January 31, 1894, at the age of eight years. She was the second child in the family and resembled M. to such an extent that the parents had only one photograph taken for the two children, inasmuch as the one represented both.

M. was born without instruments, but version was performed. She was not asphyxiated and had no convulsions following birth. She was the third child in the family. She probably had adenoid vegetations, as she experienced difficulty in nursing and in breathing through her nose. She cried for thirty-six hours after birth and is said to have "ruptured her navel and one side, and to have enlarged her head somewhat." M. was not subject to severe illness. Her sight was said to be good, but her hearing defective; this defective hearing may really have been a want of attention. She could not speak. She was right-handed and had no anomaly of gait. She began to walk before she was two years of age and had no motor paralysis. She had some of the infectious diseases (measles, whooping-cough), but never had convulsions. Her bowels were "weak;" this statement probably means that she was subject to diarrhea. The feet and hands were always cold and blistered easily. She masticated her food properly. She was said to be lacking in sensation, and would handle hot coals and hot food without uttering a sound of discomfort. She was

exceedingly active and noisy, as so many feeble-minded children are, and was destructive, heedless of danger and careless. She wet the bed as well as her day clothing, did not understand what was said to her, and could not read. The above history was obtained from the child's father, December 30, 1895.

At the time of admission to the school, October 17, 1896, she was recorded as an idiot with a large head, the circumference of which was 21 inches. Her sensation could not be accurately tested on account of her mental condition. Acuity of vision was never tested, for the same reason, but her sight was apparently good. No peculiarities of motion were noticed. The movements were not inco-ordinate and tremor and convulsions did not exist. Tendon phenomena were not obtained. No trophic disorders were observed. The child could not talk; she was unclean in her habits and noisy. Pulse was normal. Incontinence of urine existed. The number of red blood corpuscles to a cubic millimeter was 5,800,000. Hemoglobin was 74 per cent. No rigidity was detected.

After having been in the school one year she was reported as probably more troublesome and harder to manage than when admitted; although her parents seemed to think she was improved when they visited her in September, 1897.

For about a year previous to her death, M. showed very gradual emaciation, although careful physical examination revealed no special cause for it. The gait was observed to be more shambling than previously, and this was attributed to physical weakness. In February, 1899, she was reported as thin and poorly nourished.

On February 8, 1899, emaciation being extreme she was sent to the hospital, was fed upon very nourishing diet, and was kept in bed most of the time. In three weeks she had gained a little in strength and ran about the ward as usual. At this time diarrhea was troublesome for three days and was checked by simple remedies.

On March 6, 1899, at 6.20 A. M., word came from the hospital that the child seemed to be dying. Examination showed very weak and irregular heart sounds, 110 per minute; respiration 28; temperature 100 degrees F. She refused to swallow. Occasionally she moved her hands and legs. She remained in this condition, growing weaker until death resulted on the same day at 3.30 P. M.

The necropsy was performed May 9.

The body was that of a much emaciated child. The head was long, narrow and appeared rachitic. The bridge of the nose was flattened and suggested congenital syphilis. The lower front teeth were nicked like those known as Hutchinson's teeth.

The sternum was very prominent, and presented the so-called pigeon-breast. Other bones were not enlarged. The dura was partly adherent to the calvarium and the latter was removed with considerable difficulty. The membranes over the convexity of the brain presented a peculiar boggy appearance and were bluish in color. When the dura was incised on the left side about two or three ounces of dark fluid blood escaped. Two layers of new membrane could be stripped from the inner side of the dura. The layer next to the dura was exceedingly vascular, thick, edematous and of a dark red color; that more internally situated did not appear to be vascular macroscopically, but the microscope showed the presence of vessels. The innermost layer of membrane, especially on the left side, was loosely attached to the more superficial layer and the latter could be torn from the dura without difficulty, although it was distinctly adherent. The pachymeningitis extended well toward the base of the cranium on each side. The new membranes were not adherent to the pia except at the superior longitudinal fissure. The pia was very edematous and the brain was quite soft.

The brain was small and the cerebellum was very imperfectly covered by the occipital lobes. The fissures were deep and the character of fissuration was that of low-type cerebrum. The brain was not weighed at the time of removal, but the left hemisphere with the left cerebral peduncle and half of the pons, but without the cerebellum, weighed 290 grams after the tissue had been kept some time in formaline.

The liver extended upward to the fourth rib and downward three fingers' breadth below the last rib, chiefly on account of the malformation of the thorax. No scars suggestive of syphilis were found in the liver. The other viscera were examined, but nothing worthy of special note was detected.

The central nervous system was studied microscopically.

Right paracentral lobule; thionin stain: All the nerve cells were found to be much swollen (Fig. 1) and they had undergone intense chromatolysis. The nucleus was displaced to the periphery of the cell. The larger cells presented a swollen, rounded body with a pointed end, and this pointed extremity was usually the portion of the cell nearest the free surface of the cortex. In these cells the chromophilic elements were found only toward this pointed extremity. While in most of the cells the nucleus was in the pointed end, in some it occupied the other extremity of the cell body. Some of the cells were entirely deprived of chromophilic elements.

A structure, of a reticular character, stained purple with the thionin, was found in the portion of the cell deprived of

chromophilic elements (Fig. 11). This finding would correspond with the views of those who hold that a reticulum exists normally in the nerve cell, and that the chromophilic elements are merely incrustations upon the intracellular network. The cortical cells presented a striking resemblance to miniature balloons, the point at which the chromophilic elements were pre-

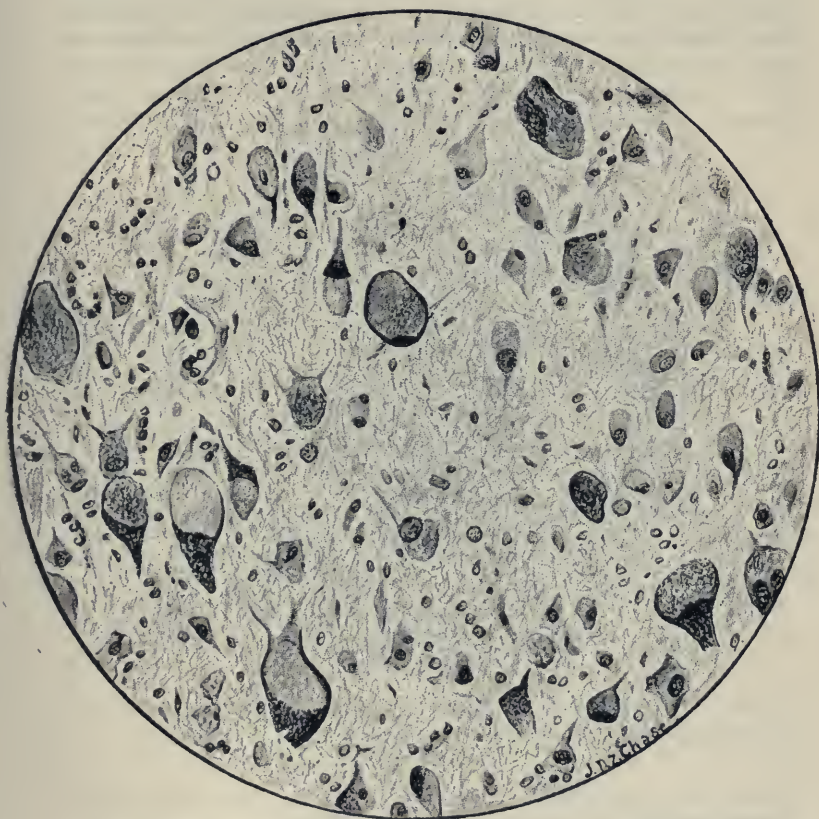


Fig. 1.—Section from the paracentral lobule. The nerve cells are swollen, and in a condition of chromatolysis.

served corresponding to the basket of the balloon. The smaller cells presented the same appearance, but the condition was especially striking in the large giant cells of the paracentral lobule. The chromophilic elements preserved in the pointed extremity did not usually present the rod-shape normal to the giant cell, but were distinctly granular. The nucleus in many

of the nerve cells stained somewhat deeply and the nucleolus stained intensely. In the apical process of some of the cells normal chromophilic elements were found.

Right paracentral lobule, Weigert's hematoxylin method:

The appearance presented by this stain was almost more striking than that by the thionin preparations. The nerve cells stained a yellowish brown. In many of the tumefied giant cells a considerable accumulation of granular matter was found that stained purplish-black with the hematoxylin, and this material was usually nearer the pointed extremity of the cell where the chromophilic elements were found by the thionin stain. In some cells this granular matter occupied the entire cell body.

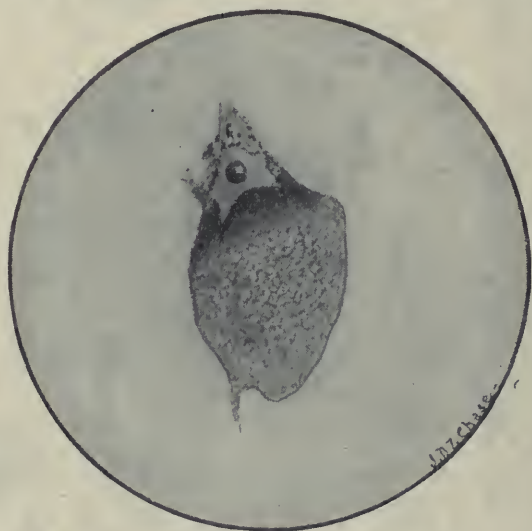


Fig. II.—One of the giant cells from the paracentral lobule under higher magnification, showing chromatolysis and a reticulum.

The nucleus could be distinguished with this stain and the nucleolus stained a deep brown or even black.

The small vessels of the cortex were widely distended with red blood corpuscles that stained intensely with the hematoxylin. The small capillaries were so numerous in the gray matter that a proliferation of these vessels in the cortex had probably occurred (Fig. III). Both gray and white matter contained many red blood corpuscles free within the tissue. Numerous red blood corpuscles were found in the perivascular spaces of certain of the vessels, and considerable collections of these red blood corpuscles were seen free in the tissues. The red blood

corpuscles free within the nervous tissues were apparently normal, and were indicative of recent infiltration. Altered blood pigment was not found within the tissue of the paracentral lobule.

Nerve cells in sections taken from the frontal lobe and posterior portion of the parietal lobe, from the cerebellum and through the bulbar nuclei exhibited similar changes to those in the paracentral lobule.

Spinal cord, cervical region: The spinal cord was hardened

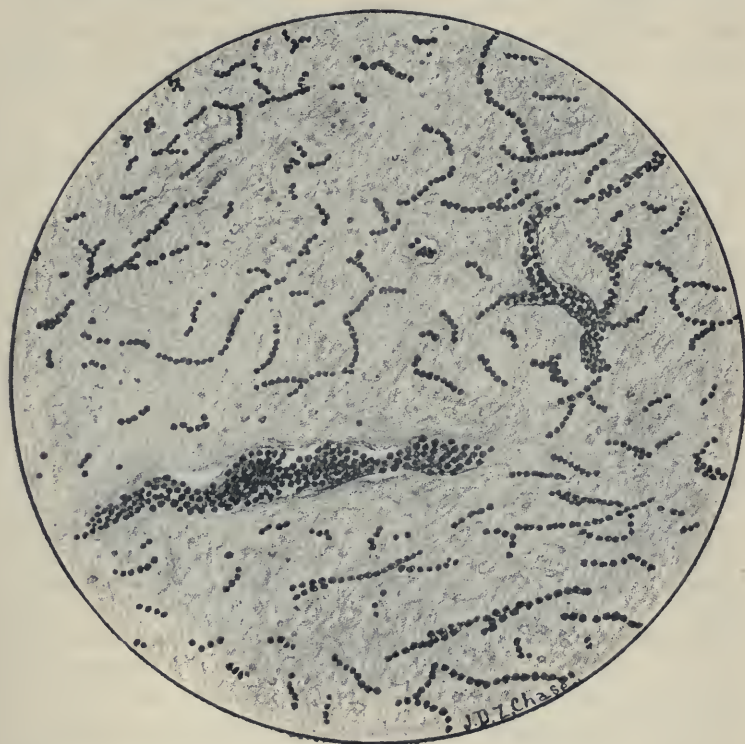


Fig. III.—Proliferation of capillaries in the cerebral cortex.

in Müller's fluid, and therefore the thionin stain could not be employed. With the Weigert hematoxylin stain, the white matter of the cord appeared to be normal. The cells of the anterior horns stained as intensely by this method as those of any other part of the central nervous system, and even by this method were seen to be greatly tumefied and often without dendritic processes. This intense coloration of the anterior

horn cells was especially striking, inasmuch as sections of the spinal cord stained by the Marchi method did not exhibit the black granules so often found in the spinal cords of the aged. The cells of the anterior horns by the Marchi method were pale yellow and entirely free from black granules. This was not due to insufficient penetration of the osmic acid, as the tissue had assumed the color always seen in normal tissue properly stained by this method.

The anterior and posterior roots by the Weigert method

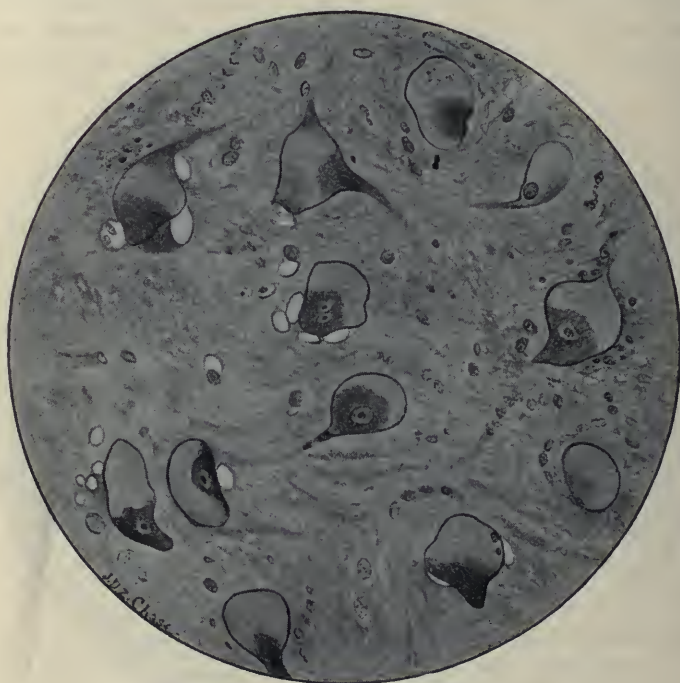


Fig. IV.—Nerve cells in the anterior horn of the spinal cord.

were apparently normal, but small black granules were seen within the intramedullary portion of the posterior and anterior roots when stained by the Marchi method. As these dots are at times found in the roots in normal tissue, we merely record the finding without asserting that the roots were degenerated.

Sections from the cervical region stained with ammonium carmine showed exactly the same changes in the anterior horn cells as above described as occurring in the paracentral lobule

and cortex generally. The cells were almost globular (Fig. IV), with a pointed extremity, and the nucleus was found in this pointed end. About the nucleus was a mass of matter that took the carmine stain intensely. The rest of the cell was a pale yellow and had no affinity for the carmine.

The small vessels of the spinal cord were intensely congested.

Lumbar region: The description of the cervical region applies equally well to the lumbar region. The cells of the columns of Clarke were as markedly affected as those of the anterior cornua, and yet no degeneration of the direct cerebellar tracts was apparent either in the Weigert or the carmine preparations.

Distinct signs of inflammation were not present in the cerebral dura. The blood vessels were engorged and in some places large accumulations of red blood corpuscles were found free within the dura, but there was no distinct round cell infiltration. The nuclei of the cells in the dura stained well, but were not as elongated as is usual in fibrous connective tissue. At one place in a section where the dura made a fold upon itself the endothelial lining was very distinct and the portion within the fold most distant from the false membrane was apparently normal. Nearer the false membrane the cell nuclei of the endothelium were more globular.

In the layer of false membrane immediately adjoining the dura were many blood vessels having walls so delicate that they were recognizable only by the presence of endothelial cells. These vessels were greatly distended and numerous red blood corpuscles were found free within the tissue. The foundation substance of this membrane was amorphous or somewhat fibrillar, and it contained many nuclei; some of which were spindle-shaped or round, but the majority were oval. They were doubtless the nuclei of fibroblasts in different stages of formation.

In the false membrane between the portion next to the dura and that most remote, the tissue seemed to be in an earlier stage of formation; the nuclei were more distinctly round or oval and the long spindle-shaped nuclei were absent.

The portion of false membrane most remote from the dura showed a further advanced formation than the portion just referred to. The tissue was more fibrillar, denser; and the nuclei, while on the whole not as elongated as those in fully formed connective tissue, were nevertheless more fusiform than those in the central layer of the false membrane. The blood vessels throughout the new-formed membrane were numerous, and it can hardly be said that they were more numerous in one

layer of the membrane than in another. Large masses of blood pigment were found within the false membrane.

The looser and more amorphous tissue of the middle layer and the character of the nuclei contained within it indicate that this portion of the membrane was of later formation, and it seems possible in the light of our experiments to be mentioned further on, that the membrane may have formed after a

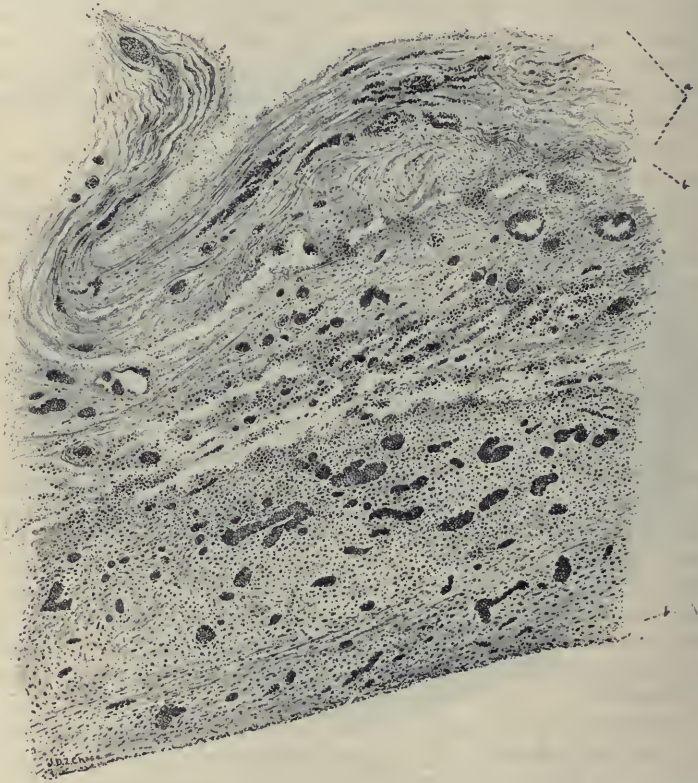


Fig. V.—The dura (*a*) is shown in the uppermost part of the drawing. Immediately next to the dura is a tissue of somewhat loose structure, and beneath this is a dense tissue rich in vessels.

subdural hemorrhage had occurred, and that the peripheral parts of the clot became organized before the central.

At the necropsy, two distinct membranes could be torn from the inner surface of the dura, and the loose tissue referred to was probably the connection between the two layers.

Groups of round cells were not found within the false membrane.

The changes in the nerve cells throughout the central nervous system, both brain and cord, were of much interest. A condition of intense chromatolysis existed and the cells were tumefied. In many cells only a few chromophilic elements remained at one portion of the cell body, and the dendritic processes had disappeared or were very indistinct. We noticed such changes in the cells of the cerebral and cerebellar cortex, in the bulbar nuclei, and in the cells of the spinal cord.

Some of the Purkinje cells of the cerebellum stained by mistake with ferricyanide of potassium and acid alcohol presented distinct small dark granules, filling the entire cell body and extending in some cases from the cell body into the surrounding tissue. The exact significance of these granules is unknown. They may possibly be the metaplasma granules of Van Gieson, or they may indicate an early stage of iron infiltration of the cell body such as Weber¹⁴ has recently described.

Red blood corpuscles stained by Weigert's hematoxylin were dark in color and the presence of the dark granules in the nerve cells of our case by this stain suggested the possibility of an infiltration of coloring matter from the blood. Weber found cerebral ganglion cells in which the cell body, except the nucleus, stained such a deep black by alum hematoxylin that they resembled the cells stained by the silver method. When the color was less intense the cells appeared to be filled with blue granules. These cells when placed in a 5-per-cent. solution of ferrocyanide of potassium and acid alcohol gave the Berlin blue color. With the Weigert hematoxylin stain the infiltrated cells were deep black, while the other cells were brownish-yellow. The siderophilous cells were always in the vicinity of diseased and bleeding vessels. Weber believed that the granules he described contained an albuminate of iron. He was unable to find an exactly analogous case in the literature and he thought that the early age of the child in his case might have rendered the nerve cells more susceptible to this infiltration.

The early age of the child in our case, the proliferation and engorgement of cortical capillaries, the free red blood corpuscles within the cerebral tissues, made the deep pigmentation of the nerve cells by the Weigert hematoxylin method seem possibly the result of infiltration by blood coloring matter. The characteristic iron reaction was not obtained even when the proper potassium salt was used. We cannot entirely dismiss from our minds the possibility that this intense coloration

¹⁴ Weber. *Monatsschrift für Psychiatrie und Neurologie*, Vol. 3, No. 6, p. 507.

tion of the nerve cells by the Weigert method was due to blood coloring matter, although we regard it as an unsettled question.

Doehle is inclined to believe that intracranial hemorrhage at birth is the cause of the pachymeningitis, and that the brain suffers at the time of the formation of the hemorrhage, although he had few findings to offer in support of the latter view. It seemed to him that cortical changes might be the cause of the deficient bodily development of these children, but the cortical changes he had in mind were those produced by the trauma that causes the hemorrhage and not the changes resulting from the presence of the membranes with the fluid contained within them—at least his words permit this interpretation.

It seems to us quite probable that an injury sufficient to produce intracranial hemorrhage may be sufficient to cause alteration of nervous tissue, possibly by pressure of the effused blood; but it is no less likely that intense proliferation of tissue on the inner surface of the dura with the production of more or less hemorrhage disturbs the nutrition of the cortical cells and leads to cellular degeneration. The presence of numerous dilated capillaries and of many red blood cells in the cortex in our case indicate that the nutrition of the cortical cells was imperfect. In addition to this, the brain was very edematous and the edema probably had some effect on the nerve cells. Systematic examination by the best methods of the cerebral cortex in cases of internal hemorrhagic pachymeningitis might frequently reveal cellular changes. Such degenerative changes occur in cases of general paralysis of the insane, chronic alcoholism, etc., and these are conditions in which internal hemorrhagic pachymeningitis is also found.

The nerve cells in our case throughout the central nervous system appear as small distended bladders. The Marchi method applied to the spinal cord shows that the nerve cells here are entirely deprived of any pigment that stains black by osmic acid. The chromatolysis therefore has not given place to pigmentation. Hirsch¹⁵ has described a similar condition as one of toxemia in his case of amaurotic family idiocy, but

¹⁵ Hirsch. *The Journal of Nervous and Mental Disease*, 1898.

the disease in our case could not be classed under amaurotic family idiocy. The child was not blind, and microscopical examination shows that the optic nerves were normal. The symptoms were not those of this disease.

It has occurred to us that the edema of the cortex might explain the vesicular appearance of the nerve cells, but the cases of uremia reported in literature do not justify this view.

Ewing¹⁶ cites the papers of Acquisto and Pusateri, Sacerdotti and Ottolenghi and Donetti. These writers examined the nerve cells in cases of experimental uremia in animals, but cellular changes such as described by us were not seen by these investigators. Ewing reports six cases of uremia in man, and in some of these the brain and pia were edematous. No cellular changes comparable with those in our case were seen by Ewing. He states also that the effects of pial edema could not be distinctly traced in the cortical cells. Ewing gives no drawings resembling the cells in our case.

It is not impossible that the cachexia was the cause of the cellular changes. Müller and Manecatide¹⁷ have examined the nerve cells of children dying within the first year of life from intestinal diseases, with and without fever. They found cellular changes in all their cases, consisting chiefly of chromatolysis and tumefaction. When the chromophilic elements were destroyed a net of fine fibrils was seen within the cell body. These changes they state resemble those of intoxication and infection, but are not characteristic; neither are the changes of intoxication and infection characteristic, according to Babes and others.

Such a condition of intense tumefaction and chromatolysis might well be supposed to lead to functional disturbance, and it may have stood in causal relation to the feeble mentality, but it seems to have had little influence over motion. Although the motor cells of cortex, medulla oblongata and cord were as much altered as the cells of any other part of the central nervous system, no marked paralysis of motion was noticed. This shows that chromatolysis is not necessarily an index of

¹⁶ Ewing, *Archives of Neurology and Psychopathology*, Vol. 1, No. 3, p. 263.

¹⁷ Müller and Manecatide, *Zeitschrift für klin. Med.*, Vol. 36, Nos. 1 and 2, p. 1.

functional disturbance. Recent investigations lead us to look to the neurofibrils as a very essential portion of the nerve cells, and these have as yet received little attention in studies of pathological processes. The importance of chromatolysis is at present a subject of debate. Nissl¹⁸ himself now acknowledges that chronic poisoning does not produce characteristic changes in the nerve cells, although subacute poisoning with maximal doses gives typical changes from each poison.

A number of writers have spoken of the absence of functional disturbance, although chromatolysis existed. The experiments of Goldscheider and Flatau¹⁹ are confirmatory of this teaching. Rothmann has recently published some experiments in compressing the abdominal aorta of dogs. In one instance the ganglion cells were much swollen and deprived of chromophilic elements, and yet the function of the posterior limbs shortly before death was almost normal.

We must refer also to Rothmann's²⁰ statements regarding the reticulum observed by him within the nerve cells. This seems to have been very similar to the network observed by us. Rothmann said the network appeared about ten hours after the compression of the abdominal aorta, and as the Nissl corpuscles disappeared. He thought it was not improbable that this was a previously existing portion of the unstained substance and that it stained in the cells undergoing degeneration, although he did not exclude the possibility of a pathological origin. He refers also to the difference of opinion regarding the normal structure of the cell. Bütschli, Held and Auerbach support the reticular theory of the "neurocytospongium," while Apáthy, Bethe, and Nissl believe that there is a fibrillary character of the cell substance.

Marinesco²¹ has reviewed the opinions of various investigators on the structure of the nerve cell, and he states that a network (*réseau*) exists within the nerve cell. The picture which he gives as figure I presents a network very like that seen by us.

¹⁸ Nissl, *Centralblatt für Nerven. u. Psych.*, Beiheft, 1898, p. 654 and *Münch. med. Wochenschrift*, No. 31, 1898.

¹⁹ Goldscheider and Flatau, *Fortschritte der Medicin*, 1897.

²⁰ Rothmann, *Neurologisches Centralblatt*, No. 2, 1899, p. 68.

²¹ Marinesco, *Archiv für Anatomie und Physiologie*, Phys. Abtheil, 1899.

It is not our intention to give a thorough review of the literature on the subject of internal hemorrhagic pachymeningitis, as it is exceedingly voluminous. The inflammatory theory seems to have been the older and Calmiel (1826) and Bayle (1826) are mentioned among the early supporters of this view; whereas Andral (1836) and Abercromby (1845) upheld the hemorrhagic theory. The latter theory seems to have found the most support until Virchow (1857) threw the weight of his influence in favor of the inflammatory origin. As Robertson²² says, a war has been waged by the supporters of these two theories for nearly seventy years, in which upward of ninety writers, mostly French, have taken part. The pendulum has begun to swing a little away from the direction given it by the writings of Virchow; and Laborde, Sperling, Huguenin, Wigglesworth, Dercum, Hoyt, and others have expressed themselves in favor of the hemorrhagic view.

The theory which Wigglesworth²³ has held might be applied to our case. Wigglesworth states that the affection is associated in overwhelming proportion with brain degeneration and atrophy, and that the main cause is the loss of support experienced by the meningeal vessels in the degenerating and wasting brain, assisted by the localized or general congestions of the meninges, so liable to occur in all forms of insanity (p. 434).

Osler,²⁴ however, believes that something more than atrophy of the brain is needed, or we should find the false membrane more commonly in cases of cerebral wasting.

We hardly feel inclined to enter into a discussion of the views held by Robertson (l. c.), viz., that the all-important element in the production of a subdural membrane is sudden lowering of the intracranial pressure, and that the effect of this is analogous to a dry-cupping of the dura mater. Theories in regard to the formation of internal hemorrhagic pachymeningitis are numerous.

We have spoken of the presence of numerous round, oval and elongated nuclei within the new membrane, but we have

²² Robertson, *The Journal of Mental Science*, Vol. XXXIX, p. 203

²³ Wigglesworth, *Brain*, 1892, Vol. XV, p. 431.

²⁴ Osler, *Journal of Nervous and Mental Disease*, 1888, p. 608.

not been able to determine the origin of these cells. It is possible, even probable, that they come in part from the white blood cells. In our experiments we found many leucocytes within the forming membrane. The view that spindle cells may develop from the white blood corpuscles has been held by many writers; it was held by Huguenin²⁵ many years ago, and Wigglesworth,²⁶ Hoyt (l. c.), and others more recently have supported the same teaching.

Sperling²⁷ found that eight days after the injection of fresh coagulable rabbit's blood between the arachnoid and the dura of the rabbit's brain, the clot began to show signs of organization. After two or three weeks the formation of a membrane was complete and in all cases he was able to observe newly formed vessels at this period. This new membrane is said by him to have corresponded in all points with that found in hemorrhagic pachymeningitis.

We have repeated the experiments of Sperling. The animals chosen were dogs and cats. Subdural hemorrhage was produced and the animals were killed at different periods following the experiment; one at three hours, one at six hours, one between six and twelve hours, one at five days, one at seven days, and one at five weeks. In other animals the under surface of the dura was painted with hydrochloric acid and the animals were permitted to live for different periods.

The technique of producing the subdural hemorrhage was as follows: The animal was etherized. A small piece of bone was removed from the left side of the skull, a needle was introduced through the dura and through the superior longitudinal sinus, and hemorrhage was produced in the subdural space on each side of the convexity of the brain. The sections were cut only from the side where the skull remained intact. In the experiments with the acid the same procedure was followed, although the dura of only one side of the brain was painted. Animals in which suppuration occurred were rejected, and only those in which no evidence of a purulent process was found were considered as suitable for our purpose.

²⁵ Huguenin, Ziemssen's Handbuch, Vol. XI, part 1, 1876.

²⁶ Wigglesworth, The Journal of Mental Science, Vol. XXXIII. 1887-1888.

²⁷ Sperling, Centralblatt für Deutsch. Med., 1871.

A large accumulation of red blood corpuscles was found beneath the dura of a cat killed three hours after the production of a subdural hemorrhage, but no distinct evidences of organization were seen.

A number of polynuclear cells were found in a subdural hemorrhage after six hours.

The evidences of the formation of a new membrane were quite distinct after five days in a subdural hemorrhage in a dog's brain. Many cells with round or oval nuclei, staining deeply with hemalum, were found in the portion of the clot immediately joining the under surface of the dura. These were evidently the nuclei of fibroblasts, although they were much thicker, shorter and more oval than the nuclei of the dura. Similar cells were found in the midst of the clot. In the portion of the clot nearest the pia a mass of amorphous matter, probably fibrin, was found, containing many of these deeply stained nuclei, much smaller and more irregularly formed than those nearest the dura. It seems, therefore, that the organization of the clot begins in the portion nearest the dura, and in the preparations from our case of pachymeningitis in a child, described in this paper, we noticed that the portion of the membrane next to the dura presented a more advanced degree of development.

The new membrane caused by a subdural clot in a dog was very distinctly formed after five weeks. The red blood corpuscles in the clot showed alteration of structure, and beneath these in the portion of the clot nearest the pia a new membrane was found rich in cells. The nuclei of most of these cells were elongated; some were oval or irregularly formed. In one field a distinct capillary could be seen in the new membrane; its endothelial cells were very visible and it contained red blood corpuscles. The great quantity of nuclei in this new membrane were indicative of a very active process.

The sections of the subdural clot from animals permitted to live a few weeks after the formation of a subdural hemorrhage furnish indisputable evidence that a subdural hemorrhage may be *one* of the causes of the formation of a subdural membrane.

An unusual number of cells with elongated nuclei were found on the under surface of a cat's dura on which hydro-

chloric acid had been used and the animal permitted to live three weeks, but there was no distinct evidence of a newly formed membrane. Even after seven weeks the evidences of a newly formed membrane were not more distinct than in the specimen removed after three weeks. We would conclude from this, that if irritation is capable of producing a false membrane, it must be an irritation that is constant.

The experiments of Goodall²⁸ seem also to indicate that experimental irritation of the cortex does not produce a false membrane of the dura. He applied sp. vin. gallic. or diluted cantharidin to the cerebral cortex of rabbits through an incision of the dura, and in five animals no inflammatory exudate or naked-eye evidence of inflammation was found; in a sixth a *hemorrhage* was produced in some way and a pseudo-membrane formed.

DISCUSSION.

Dr. E. D. Fisher thought that the early idea that hemorrhagic pachymeningitis was rare was due, as Osler said, to observing the wrong class of cases. In any almshouse this condition would be found very prevalent. Some years ago the question came up in the New York Pathological Society, and he took occasion at that time to show a number of cases of this disease. He did not quite understand the etiology of the case reported by Drs. Spiller and McCarthy, and asked for further information.

Dr. D. J. McCarthy replied that the etiology of the case was rather obscure, there being evidence both of rachitis and syphilis. Diarrhea existed for a short time before death, but that probably had no causative relation to the membrane itself; the etiological factors were syphilis and rachitis.

Dr. McCarthy stated that the statistics show that 2.7 per cent. of the entire number of cases of pachymeningitis occur during the first year of life.

Dr. W. G. Spiller said that cases of internal hemorrhagic pachymeningitis are not very uncommon in the insane, as Dr. Fisher had stated, but they occur much less frequently in childhood, and he believed that no case in a child so old as nine years had been published. In the case just reported, the flat bridge of the nose and the Hutchinson teeth were indicative of syphilis; and the so-called pigeon-breast was a sign of rachitis.

²⁸ Goodall, The Journal of Mental Science, Vol. XXXVIII, p. 397.

SENSORY DISTURBANCES IN EPILEPSY AND HYSTERIA.¹

By E. D. FISHER, M.D.

Dr. Fisher said that in both conditions hemianesthesia might be present, but it was more frequent in hysteria. General or bilateral anesthesia was not uncommon. He found this condition often existing independently of any seizures, and reported two cases. The first was that of a woman aged seventeen years. She had a history of true epileptic seizures since her first menstrual period three years previously. Examination showed complete anesthesia of all forms of sensation over the middle of the forehead, both shoulders, the dorsal surface of both hands, and the extensor surface of forearms. The second case was that of a woman twenty-four years of age. A diagnosis of hysteria was made. There was anesthesia of both shoulders, dorsal surface of the hands, and extensor surface of the forearms. In both cases the condition was permanent, and probably of cerebral origin. It was evidently not of peripheral origin. In view of these cases and the literature on the subject, it would be well to examine for sensory symptoms in all cases of epilepsy.

DISCUSSION.

Dr. P. C. Knapp said he had occasionally noted the presence of the sensory disturbance, of which Dr. Fisher spoke, in epileptics, where it seemed pretty clear that the attacks were of epileptic character, and not hysterical, and he thought we should probably find slight sensory disturbances oftener if we made careful comparative tests of the sensibilities of the two sides.

Dr. E. D. Fisher thought that when we examine the literature and find that in case after case of epilepsy reported by a large number of observers these permanent, and also transitory, conditions of anesthesia have been found, we must allow ourselves to accept the diagnosis of epilepsy in such cases, or we cannot come to any conclusion whatever.

He thought probably we are dealing with some central nervous disturbance aside from what we may call cases of chorea, epilepsy, or hysteria, that may lead us to think of a diffuse lesion, such as might manifest itself later as a case of

¹Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

multiple sclerosis. Thompson and Oppenheim are inclined to ascribe it all to cerebral disease, but again they have had cases similar to these Dr. Fisher had described, and the ultimate history has been that they turned out to be multiple sclerosis. Where the early diagnosis is a difficult one to make at the time, definite symptoms of multiple sclerosis may show later that we are dealing with a disease of a degenerative type.

PURULENT ENCEPHALITIS AND CEREBRAL ABSCESS
IN THE NEW-BORN, RESULTING FROM INFEC-
TION THROUGH THE UMBILICUS.¹

BY GUY HINSDALE, M.D.

The mother of the patient had had a painful pregnancy, and after labor lasting forty hours pus from the uterus followed the placenta. The child died on the thirteenth day, after symptoms of meningitis. All the surfaces of the cerebrum and cerebellum were covered with a fibro-purulent exudate; a pus cavity was in the left anterior lobe, and a large hemorrhage in the right. There were innumerable hemorrhages throughout the cortex, with destruction of the infiltrated nervous tissue. The microscopical study was made by Dr. Joseph Sailer; the bacteriological examinations by Dr. S. S. Kneass. The bacteriological examination showed a pathogenic organism corresponding in its description with the colon group, probably the bacillus coli immobilis.

¹Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

October 3, 1899.

The President, Dr. Frederick Peterson, in the chair.

CEREBELLAR TUMOR.

Dr. W. B. Noyes presented a boy of five years who had been well until last June. At that time he had an obscure illness diagnosed as an auto-intoxication. He remained unconscious for two days. During July he had been in fair condition. In August he was noticed to stagger when walking. He could move his legs when sitting, but had absolute ataxia. A slightly spastic condition of the legs interfered with properly obtaining the knee-jerk. Ankle-clonus could be occasionally elicited. No sensory disturbances of any sort existed. His mental condition was becoming slowly worse. Examination of the eyes showed papillitis, with hemorrhages over the disk. The pupils were dilated and did not respond to light. He had neither headache nor vomiting. The case was presented as probably one of cerebellar tumor. The speaker said that it was not impossible that the sickness which occurred last June was a meningitis; hydrocephalus was also possible.

Dr. George W. Jacoby said that he had been impressed with the general laxness of the boy and the bending forward of the thorax. This general loss of muscular tone made him believe the diagnosis of cerebellar tumor to be correct.

Dr. Hughes, of St. Louis, said that the case suggested cerebellar tumor, but it seemed to him not improbable that cerebrospinal meningitis had existed, and that the present condition was the result of this meningitis.

Dr. Edward D. Fisher inclined to the view that the case was one of cerebellar tumor, although admitting the force of the statement of the last speaker.

Dr. Noyes thought that the condition of the eyes alone was sufficient to settle the question of the diagnosis. He had been unable to find the report of a case of hydrocephalus producing optic neuritis, although it might cause optic atrophy when very far advanced. The case was evidently progressing rather rapidly now.

BRACHIAL PLEXUS PARALYSIS FROM PRESSURE.

Dr. William M. Leszynsky presented a young woman having a brachial plexus paralysis affecting each arm, the result of pressure. The biceps group was paralyzed; the extensors and the deltoid were extremely weak, and atrophy was observed about the shoulder joint. No sensory symptoms were detected. The faradic irritability was lost in the nerves and muscles. The same condition, although to a less degree, was present on

the right side. The paralysis had existed three months. The etiology in this case was interesting. The woman had been operated upon about three months ago for an abdominal growth. Immediately afterward a paralysis developed, undoubtedly from the pressure exerted for about one hour and a half by an apparatus used for support. Dr. Leszynsky said he had seen similar cases of pressure paralysis, but in this instance the paralysis had been so severe, and had lasted so long, that it brought up the interesting question as to whether or not the surgeon might be liable to be sued for damages. On inquiry he had learned that this form of paralysis was not at all uncommon at the present time in surgical practice, yet it was entirely avoidable. In an article by Dr. Garrigues several cases had been reported in which the paralysis had lasted for a year or more, and had been followed by atrophy. The apparatus referred to usually made pressure between the clavicle and the first rib.

Dr. Fisher said that these cases of pressure paralysis were quite commonly observed after operations, as for example, after operations for appendicitis; but he had never before seen a case in which both sides had been involved. Negligence in attending to the position of the arm during operation was often responsible for such paralysis.

Dr. Fraenkel said that while many cases of post-operative paralysis had been reported, it should be remembered that in some the paralysis had been ascribed to the ether, although in others the influence of pressure had been definitely shown. This made it difficult to hold the surgeon liable in many instances.

Dr. G. W. Jacoby said that the various cases had been recently tabulated and the conclusion reached that all the paralyses could only be explained by pressure. The consensus of opinion at the present time seemed to be that the ether could not give rise to this form of paralysis. Surgical assistants were often very negligent about these matters. For example, he had found an assistant making hypodermic injections of ether and brandy in a case of collapse, directly over the musculospiral nerve, and the ensuing paralysis he would have found difficulty in explaining had he not seen these injections being made. Again, he had known it to be positively asserted that there had been no pressure, and yet he had personally seen the etherizer resting his elbows heavily over the patient's clavicles.

Dr. Frederick Peterson said that he had seen even the peroneal nerve affected in this way after ether narcosis. In every case that he had investigated he had found that there had been a possibility of the paralysis having been produced by pressure. The question was of great importance, for he believed it would be perfectly possible for such a paralyzed patient to collect damages from the surgeon on account of such negligence. Nearly all the pressure paralyses occurring at night developed during alcoholic sleep, and he had come to look upon these as occurring in nerves which had been rendered especially vulnerable by ether or alcohol, or, as suggested by Dr. G. W. Jacoby, the inhalation of carbonic oxide in close sleeping-rooms. In cases of musculospiral palsy the nerve was generally pressed upon when the patient slept with the arm raised, and the hand under the back of the head.

Dr. Hughes said that the paralyses following the comatose state

in epilepsy exhibited local peripheral expressions. Neurologists had been accustomed to consider these as of central origin. His own opinion was that ether was a factor which could not be ignored—that it was a toxic factor. He had not yet been convinced that these cases were exclusively peripheral paralyses, and, owing to this lack of unanimity among neurologists regarding the causation of these cases, their medico-legal aspect assumed grave importance. Many of these patients recovered, as did the patients with epileptic paralyses, and he was inclined to think that the toxic condition of the brain found associated with etherization was an important etiological factor.

Dr. Leszynsky said that he could not agree with the views of the previous speaker regarding the effect of etherization. When a healthy person was etherized it could hardly be said that the toxic condition could so soon be responsible for the paralysis; it was rather a case of extreme pressure made during unconsciousness. In his own mind there was no question that all of these cases were directly due to pressure made during unconsciousness. Ether, like other stimulants, might give rise to cerebral hemorrhage, or induce hemiplegia of the hysterical type. If these cases were due to a lesion in the anterior horn they would not all recover, as occurred invariably in the class of cases under discussion.

THE CENTRAL NERVOUS SYSTEM IN ACUTE MALARIAL INFECTION.

Dr. James Ewing read this paper. He introduced the subject by reporting the three following cases:

Case I.—*Estivo-autumnal malaria*, with marked cerebral symptoms and infection with a single, well-defined group of parasites. The patient was a soldier who had just returned from Cuba. The stupor lasted thirty-six hours prior to death. Two days before death many ring-shaped organisms in the blood had been found. The stupor began twelve hours after sporulation occurred. At the autopsy the viscera showed the lesions of severe malarial infection. The capillaries of the cerebrum, cerebellum, medulla and cervical cord contained a large number of cells harboring parasites. Many capillaries were occluded by thrombi. The ganglion cells showed everywhere a reduction in size, irregularity and splitting or loss of the chromatic bodies. Death was due to the massing of the parasites in the capillaries. The case was a good example of comatose malaria of the cerebral type.

Case II.—This patient had chills on alternate days for several days, and, on admission, had a pulse of 110, and was much prostrated. Marked insomnia and mild delirium existed for awhile; then the delirium became still more marked, and finally coma developed, lasting for some time before death. No evidence that the coma was uremic was obtained. The temperature reached 108° F. shortly before death. On October 12 the blood contained an enormous number of ovoidal, spherical and crescentic bodies. On October 25, or eight hours before death, very few crescents could be seen. None of the younger forms were found. The autopsy showed moderate edema of the brain, the basal vessels normal, and very few parasites present. The deposit of pigment in the brain was slight. Throughout the cortex the cells showed no marked change further than a uniform diminution in the chromatic substance. In many of the pericellular lymphocytes were peculiar structures—elongated fibers or rods with tapering ends—of undetermined nature. Similar deposits were found in other cases of malaria, and in one case of tubercular meningitis. The chief feature of

this case was the prolonged delirium and coma, yet the post-mortem examination showed far less change in the kidney than was usually seen in profound malarial infection.

Case III.—This was a case of fatal malaria associated with hemoglobinuria. The man had contracted malaria in Santiago in July, 1898. He was comatose, extremely emaciated, excessively anemic and moderately jaundiced. He was at Camp Wikoff for three days prior to death. The dura and pia were distinctly jaundiced. The cerebrum, cerebellum and medulla showed the usual injection of vessels with blood, but the vast majority of the vessels were free from the parasites. An abundant deposit of hematoidin in the kidneys was interesting.

Commenting upon the foregoing cases, the speaker said that when the brain cortex was markedly brownish it indicated usually the presence of a large number of parasites, but this was not an invariable rule. The majority of cases of comatose malaria did not exhibit the massing of the parasites in the brain. Of eight cases in which he had examined the brain after death in none was there a distinct brownish discoloration of the brain. Hemorrhages had been found in some cases, as had also a moderate degree of edema. Usually the parasites were uniformly distributed in the brain and cord, but a case had been reported in which they were localized in the medulla. The number of these parasites was often enormous, and complete occlusion of the vessels was not at all uncommon. While most of the fixed pigment was found in the endothelium, the parasites were rarely seen in the endothelial cells. It was probably safe to refer the cerebral symptoms observed in these severe cases of malarial infection to the general condition of the obstructed circulation. The ganglion cells, in cases of comatose malaria, had been studied, and the changes found to consist chiefly in the various degrees of chromatolysis. The dendrites were usually involved before the cell body. The ganglion cells appeared to suffer less than in the average case of typhoid fever. The mere presence of the parasites in the tissues seemed to exert no bad influence, except in a mechanical way. In one of the cases reported in the paper the deepening of the coma could be apparently connected with the progressive filling of the capillaries with the parasites, and the formation of thrombi. In the other two cases the parasites were few and the pigment scanty. In both of these cases the malarial infection was exceedingly severe. These cases showed that the coma of malaria was not always referable to the presence of the cerebral parasites. Of the writer's sixty-four cases of malarial coma reported there were five simple tertian infections. In the estivo-autumnal cases, with crescents only in the blood, there were thirty-four instances from the Montauk camp. Coma appeared to be rather frequent in cases showing only crescents in the blood. Crescents did not exhibit such a tendency to unequal

distribution as did the fertile forms. From the condition of the viscera found in the second and third cases reported—and this variety occurred especially in cachectic individuals in whom the disease had existed for some weeks—it seemed probable that the coma was the result of the general and profound malarial infection. The writer did not believe that much importance should be attached at the present time to the condition of the ganglion cells in malaria. When the coma resulted from the massing of the young parasites in the brain, it was generally gradual in onset, and the prognosis was very unfavorable. In some cases the coma developed suddenly, and was more amenable to appropriate treatment. The general clinical character of these cases indicated that an embolic process was concerned in their causation. Of eleven cases of the first class, reported by the writer, ten proved fatal. To summarize: The three anatomical conditions were: 1. The mechanical obstruction of the cerebral capillaries by large numbers of young parasites; 2. An embolic process causing an occlusion of some vessels (usually temporary) by parasites or pigmented leucocytes, and 3. A general toxemia, such as might occur in other infectious diseases, the blood showing few, if any, autumnal rings.

Dr. W. B. Noyes said that two years ago he had seen a case showing coma, stertorous respiration, partial paralysis and irresponsive pupils. The blood taken from the spleen at autopsy showed the presence of pigmented leucocytes, parasites of the estivo-autumnal variety. Sections from every part of the brain, without staining, showed that the walls of the capillaries were infiltrated with pigment. The absence of the parasites and the enormous amount of pigment were especially noteworthy. The usual lesions of pernicious malarial fever were found in the other organs of the body. In this case the diagnosis during life had been cerebral hemorrhage by almost every physician who had seen the case—certainly the clinical appearances were the usual ones found in cerebral hemorrhage.

Dr. Lewis A. Conner thought the jaundice might explain some of the comas that were otherwise inexplicable. It was surprising sometimes how an apparently moderate degree of jaundice might be associated with very marked cerebral symptoms.

Dr. Fraenkel said that he looked upon most of the nervous consequences of malaria as due to toxemia.

Dr. Hughes said that the paper was very satisfactory in that it explained certain old and well-known clinical facts. Williamson, of England, had been one of the first authors to refer to the effect of malaria on the nervous system.

Dr. William Hirsch reported a case of a gentleman sent to him from the South about one year ago, with a diagnosis of spinal tumor. The symptoms had been atrophy of the muscles of the left shoulder girdle, associated with intense pain. The patellar reflexes were exaggerated. After having had the case under observation for some time, and studying the previous history very carefully, he had become convinced that this was not the correct diagnosis. The patient gave a history of having had a somewhat similar attack some time before, which had been recovered from. While under observation the man had developed a typically malarial temperature, and this had been asso-

ciated with splenic enlargement. Examination of the blood at that time had been negative. After about six weeks of anti-malarial treatment recovery had been complete. A very peculiar symptom in this case was a decidedly tender swelling over the sternum. This had disappeared as the patient had improved. The man, against advice, had returned to his home in the South, and when seen again, a few weeks ago, had been in a worse condition than the year previous. The left clavicle then exhibited a tender swelling, like that observed previously over the sternum. There were similar swellings over one ulna and on one portion of the skull. He was now improving rapidly under anti-malarial treatment. The case was reported because of this peculiar affection of the bones.

Dr. Teller, of Memphis, said that he had practised in a district in which malaria was exceedingly common and very malignant. In his neighborhood these peculiar neurotic manifestations of malaria were so common as to make the physicians constantly on the lookout for them. The cases of cerebral congestion were all too common, and very frequently proved fatal. The physicians of his locality looked upon the disease as a profound toxemia.

Dr. Ewing said that he had been especially interested in the remarks of one of the speakers concerning cases exhibiting jaundice. These cases commonly showed at autopsy profound and peculiar changes in the liver. In the case reported by Dr. Hirsch there were certain features that seemed to him to point to syphilitic infection. Dr. Ewing thought that the medical profession was too prone to jump to the conclusion that a disease was malarial because it seemed to respond to the administration of quinine. A very large number of nervous conditions reported, that had been referred to malaria by the older writers, should be excluded at the present time, because they had not been properly authenticated according to modern notions.

222. ON A FORM OF NERVE TERMINATION IN THE CENTRAL NERVOUS SYSTEM, DEMONSTRATED BY METHYLENE BLUE. W. A. Turner and W. Hunter (Brain, 22, 1899, p. 123).

The authors used a method adapted from Ehrlich, as follows: Small quantities of a saturated solution of methylene blue were injected subcutaneously at intervals of from fifteen to twenty minutes until the animal died. The central nervous organs were then rapidly removed, sliced into convenient blocks, and placed in a solution of ammonium molybdate for purposes of fixation. After washing, hardening and embedding, the blocks are cut as usual and mounted with a coverglass. A number of nervous structures were examined from a variety of animals, and the results obtained are given in résumé thus:

1. In the majority of cases a cellulipetal fiber may be traced to, and found to break up over, the cell body and base of the protoplasmic processes. In no case has the cellulipetal fiber been found to break up previous to reaching the cell body.

2. This lattice work is most clearly defined over that portion of the cell, namely, the root of the axis cylinder process, which is deficient in the so-called Nissl bodies.

3. It is also clearly seen over cells whose general staining is faint.

4. When the cell is rendered indistinct by changes in the focus, the lattice work is thrown into clearer perspective.

5. As best seen in the nucleus of the trapezium, the ending may be apparent without the cell. The paper is illustrated by some wretched micro-photographs and some schematic pen drawings. JELLIFFE.

CHICAGO NEUROLOGICAL SOCIETY.

April 25, 1899.

The President, Dr. Richard Dewey, in the chair.

Dr. M. L. Goodkind presented a patient with multiple sclerosis, twenty-three years of age, of Russian parentage, and with negative family history. When seven years of age he was lost in a blizzard and almost succumbed to exposure. Shortly afterward he began to exhibit the first symptoms of the malady, being easily fatigued and having some difficulty in walking. He also began to fall behind in his school work, although he had previously maintained a good standing, and had occasional attacks of headache and dizziness. At the age of thirteen he had an apoplectoid attack affecting the right side, and when fifteen years old ptosis of the right lid appeared and lasted six months. Unsteadiness in gait was then added and gradually became more marked, the limbs as well as the trunk and head being thrown into violent oscillations on voluntary effort. Speech became defective, but he disclosed no bladder, sensory or visceral disturbance, was well nourished and of fair size. At the present time the stiffness and inco-ordination of the legs are very pronounced, and the same symptoms are easily elicited in the upper extremities, more readily upon the left side. Nystagmus, lateral, vertical and rotary, is easily demonstrated, the pupils are normal, but the fundi show secondary atrophy with a peculiar discoloration of the disks.

The remainder of the evening was devoted to an informal discussion on brain tumor, which was opened by Dr. Henry M. Lyman. He said that in approaching the subject of brain tumor he was forcibly reminded of a remark which occurs in one of Hughlings Jackson's lectures: "It is a great misfortune, but it is a fact, that the more experience a man gets in the matter of diseases of the brain, the less certain he feels in his diagnosis." Much of this uncertainty with regard to cerebral tumors depends upon the various origin, nature and seat of the new growths and their encroachment upon the various organs of which the brain is acknowledged to be composed. In many cases the existence of a brain tumor is not revealed by any sensory, motor or focal symptoms, and it may exist for a considerable time and result fatally with nothing to indicate its presence in the brain. Dr. Bristow, for instance, had reported three such cases in which the only symptoms were of a hysterical character.

Dr. Lyman related the case of a man who had consulted him because he had had convulsions during the previous night. He had some difficulty in moving the left arm, his condition otherwise being normal. After this the arm gradually became

weaker, the weakness then extending to the leg, until he became entirely disabled. He then passed from observation, but it was learned later that he had been operated upon for a supposed mastoid abscess because of failure in hearing. Additional symptoms were headache, vertigo, vomiting and convulsions, with total left hemiplegia. The patient finally died, the autopsy revealing a large sarcoma of the right central convolution.

Another case of interest was that of a boy, five years of age, who developed headache, progressive loss of vision, vomiting and, especially, great unsteadiness of gait, so that he was obliged to stand and walk with the feet wide apart. The post-mortem examination showed a large osteosarcoma springing from the base of the cranium, just back of the chiasm. Dr. Lyman said that in cases like the foregoing the diagnosis was easy, but in some others the symptoms were exceedingly puzzling and obscure.

Dr. A. F. Lemke exhibited the brain of a patient who had been admitted to the Illinois Eastern Hospital for the Insane, with a diagnosis of general paralysis. It was only the routine ophthalmoscopic examination that had led to the accidental discovery of double choked disk, and hence to a change in the diagnosis. The patient had had several attacks of vertigo and vomiting, vision had been disturbed for some time, and she complained of frontal headache. There were no focal symptoms, although she had a peculiar twitching of both sides of the face. She died after a series of convulsions, having been in the institution for a year and a half. At the necropsy a sarcoma about the size of a small orange in the left frontal lobe was found. The growth contained much fibrous tissue, was large enough to make, by pressure, quite an excavation in the right frontal lobe, and was enucleated with great ease. Could the tumor have been accurately located, it might have been removed by an operation, but the removal would have left a considerable cavity, and it is doubtful whether the patient's mental condition would have been improved.

Dr. Hugh T. Patrick had examined Dr. Lemke's patient when she had optic atrophy following the optic neuritis. He had been unable to discover any focal symptoms. He thought that in many instances the inco-ordination occurring in frontal tumor was not due, as had been asserted, to polar pressure on the cerebellum, but to sensorimotor disturbance due to the increased pressure on the cerebrum.

Dr. Patrick then demonstrated three specimens of brain tumor, each case illustrating different clinical peculiarities of cerebral growths.

The first case was that of a girl of sixteen, and the first

symptoms were three general and very prolonged convulsions. Some weeks later she began to have focal fits, which were at first confined to the left arm, but afterward involved the face. On account of the length, violence and peculiar characteristics of the first convulsions, and because of the peculiar features of the Jacksonian spasms and the fact that she never lost consciousness during them, and because of the occurrence of very irregular and erratic headaches, she was supposed to be suffering from hysteria. When seen by Dr. Patrick she gave all the typical symptoms pointing to a tumor in or near the right arm center, but probably subcortical, and operation was advised. A large opening was made over the arm area, but on account of the enormous intracranial pressure the surgeon deemed it inadvisable to open the dura. Nothing abnormal could be discovered through this membrane. After the operation the patient improved and was almost entirely relieved for a period of three months, the intense optic neuritis almost completely disappearing as well as the headache, strabismus and hemiparesis. After a time all the previous symptoms returned, and she was again relieved for nearly three months by full doses of mercury and iodide of potassium. Again she became very much worse; a surgeon finally aspirated (supposedly the lateral ventricle or a cyst) a number of times, and finally injected the same cavity with iodoform emulsion, the patient dying a few hours later. At the post-mortem examination a large cystic glioma was found in the arm center, the cortex covering the growth being as thin as paper.

In the next case, that of a middle-aged woman, the first symptom had been a typical Jacksonian fit affecting the left arm. She soon had another convulsion, attended with loss of consciousness. After this the attacks varied in character, sometimes resembling an ordinary attack of petit mal, sometimes beginning locally on the left side and sometimes on the right. Other symptoms developed very slowly, the more prominent ones being mental hebetude, general nervousness and depression not unlike melancholia. The diagnosis of tumor was made, and the location was thought to be in the parietal lobe on the right side, back of the arm center. She finally died, and a tumor was found about as large as the last joint of a man's thumb, subcortical, in the right leg center, posterior to the fissure of Rolando. Could the growth have been accurately located during life and found by the knife, it is very doubtful if it could have been recognized *intra vitam*, even if cut into. The growth was an infiltrating glioma, and so closely resembled in appearance the normal gray matter of the cortex as to make a diagnosis of tumor somewhat doubtful, even when the growth was cut into after removal of the brain. It looked

somewhat like a bruise, but the microscope revealed the typical structure of glioma. The case was of particular interest because of the inconsistency of the symptoms with the size and location of the tumor.

The third brain shown was that of a middle-aged man, and the early part of the history resembled very much that of a general parietic. On closer study, however, it was thought that the apparent mental failure, as well as the apparent failure of vision, was due to sensory aphasia, as at the time of the examination the patient showed well-marked word-deafness and word-blindness as well as homonymous hemianopia. Wernicke's hemiopic reaction was distinctly demonstrated. Because the motor aphasia seemed to be more pronounced than the sensory aphasia, and because of the presence of Wernicke's sign, the tumor was located deep in the temporo-sphenoidal lobe, but the autopsy revealed a large, subcortical glioma in the parietal lobe, extending back as far as the parieto-occipital fissure, and forward almost to the operculum.

Dr. Sidney Kuh reported first a case observed in Vierordt's clinic which was supposed to be one of carcinoma of the liver, until the patient suddenly became comatose and died, when a tumor the size of a hen's egg was found in the cerebellum. It had destroyed nearly all of the vermis superior, and yet had produced no symptoms.

He also mentioned the case of a boy of thirteen who had typical attacks of mental epilepsy. Some time after the examination he developed symptoms of brain tumor, from which he died.

A third case was that of a boy of four years, the two interesting points in the case being, first, the fact that the sutures of the skull opened in consequence of the growth and the head became very much enlarged, and, second, the fact that the child had typical Jacksonian epilepsy confined to the right side of the body, the necropsy showing a large tumor, which had destroyed the greater portion of the right hemisphere of the brain. Dr. Kuh explained the convulsions upon the same side as the tumor by supposing that the growth had pressed upon and irritated the other (left) hemisphere.

Referring to a statement of Dr. Patrick, that in one of his cases it was difficult to see how such a small tumor had produced all the symptoms and finally caused death, he mentioned a case which he had seen in which the patient had suffered from the general symptoms of brain tumor and in which, at the autopsy, nothing abnormal was discovered except a cyst no larger than a small cherry in one hemisphere of the cerebellum, and the pathologist who made the examination was of the opinion that the cyst was congenital.

The speaker considered the appearance of headache to be of importance in determining the operability of tumor. If the headache occurs as a late symptom, it is probable that the tumor is deep-seated; when it occurs early, the growth is probably cortical or subcortical. In one case, although Jacksonian epilepsy was present, an operation was discouraged because of the late appearance of headache, and the post-mortem examination justified the advice, as the tumor was deep-seated.

Dr. Archibald Church called attention to a condition which he believed to be comparatively rare, and which produced the symptoms of brain tumor. He had seen two cases, and in each instance an operation revealed what seemed to be an aneurism in or upon the cortex. He was of the opinion that the condition might sometimes be determined by auscultation, and probably also by means of the X rays.

Dr. Daniel R. Brower related the case of a man fifty-five years of age who had Jacksonian epilepsy and the classical symptoms of intracranial tumor. Under the administration of iodide of potassium he temporarily improved, but the paroxysms returned and he was operated upon for removal of the growth. No tumor was found. As he had for some years had considerable trouble with a suppurating ear, the mastoid was operated upon to evacuate a possible abscess, but none was found. A few days afterward the man died, and the autopsy revealed a subcortical tumor the size of a walnut beneath the arm center, and an abscess in the mastoid cells. He had never seen any but temporary benefit from surgical interference in tumors of the brain.

Dr. James B. Herrick mentioned a case of frontal tumor in which the symptoms were principally mental, the condition being one of melancholia and despondency, in consequence of which the patient attempted suicide by cutting his throat with a razor. Dr. Herrick also mentioned the great difficulties of diagnosis in cases of multiple tumors of the meninges, and related one case in which he and other competent observers had supposed the case to be one of tuberculous meningitis. In fact, the case was exhibited in the clinic as a typical one of this disease, the autopsy revealing diffuse sarcomatosis affecting the pia matter of the brain and cord. In reviewing the case afterward, he thought that two facts should have aroused suspicion, namely, the long continuance of the disease (ten or twelve weeks) and the very slight elevation of temperature, seldom as high as 100 degrees.

Dr. Sanger Brown thought that we often lose sight of the fact that in cases of brain tumor many of the severest symptoms are actually neurasthenic, and not, as is too frequently believed, focal. For instance, patients with tumor are very likely to

awake at from three to five o'clock in the morning with a profound and insufferable headache which is entirely comparable to that of neurasthenia. Dr. Brown believed that this headache is caused by the depressing effect of the growth upon the vital portions of the encephalon, rather than by irritation of the meninges or of any of the cerebral tissues. He also criticised the doctrine that pressure or irritation of the growth in the brain is always the cause of existing optic neuritis.

Dr. Richard Dewey spoke briefly of the mental symptoms of brain tumor, which have received little systematic attention. It cannot be said that any typical form of mental disturbance belongs to brain tumor, the most frequent being dullness, apathy, semi-stupor, at times a good deal of emotional disturbance as the result of the irritation and pain, but in rare cases attacks very much like mania. He further called attention to the fact that a very considerable number of cases of brain tumor present in a general way the symptoms of general paresis, and are mistaken for that disease.

223. A NOTE ON THE INFLUENCE OF MATERNAL INEBRIETY ON THE OFFSPRING. W. C. Sullivan (Journal of Mental Science, 45, 1899, p. 489).

The defective classes, it is well known, are largely recruited from among those of alcoholic habits and from the offspring of such alcoholics. Careful studies upon this question have been many, though this is true that but few observations have ever been made on the normal members of an alcoholic family tree. The present study follows in detail the life histories of a number of alcoholic families, from which the author draws a number of inferences.

Maternal inebriety is a condition peculiarly unfavorable to the vitality and to the normal development of the offspring. Its gravity in this respect is considerably greater than that of paternal alcoholism.

While its influence, particularly as measured by the test of infant mortality, appears to be exercised in considerable degree indirectly through deterioration of the milieu, a large part also depends on the primary action of the poison. This is evidenced by the tendency to still-births and abortions, by the high rate of epilepsy.

This primary influence of alcohol is due in part to the permanent effects of the poison on the maternal organism, inducing a transmissible degenerate condition, in part to a direct toxic action on the embryo, owing to continued excesses during pregnancy and lactation.

Under the modes of influence the normal tendency of the family with alcoholic maternity is toward a type the inverse of the syphilitic family; that is, the first born are normal, then come more or less defective children who live beyond infancy, then early deaths, still-births, and, finally, abortions.

JELLIFFE.

Periscope.

PATHOLOGY.

224. EIN FALL VON TABES DORSALIS INCIPIENS MIT GUMMÖSER ERKRANKUNG DER HIRNSUBSTANZ (A Case of Tabes Dorsalis Incipiens with Gumma of the Brain). Nonne (Berl. klin. Woch., Vol. 36, 1899, Nos. 15, 16, 17).

Nonne reports a case of tabes running an atypical course and presenting several interesting features. Besides headaches, lancinating pains, ataxia, etc., there was a unilateral absence of the knee-jerk, which subsequently returned; unilateral slowing of contraction of the pupil to light; partial optic atrophy not progressive; and finally, epileptiform convulsions, dementia and general spastic paresis. He assumed an atypical tabes with arterio-sclerosis—syphilitic (?)—of the cerebral vessels, which led to an area of softening affecting the motor tracts. The autopsy revealed an early posterior sclerosis with involvement of the posterior roots (not complete); a gumma of the left lenticular nucleus, and an area of softening of the right lenticular nucleus. There was also a sclerotic degeneration of the crossed pyramidal tracts. He explains the return of the knee-jerk on the right side, not by any specific cord change, but by the presence of the cerebral new growth, which, by destroying the effect of cerebral inhibition over the lower reflex arc, permits a motor response to a much lighter sensory stimulus than would normally be required. He, however, does not exclude the possibility of secondary degeneration in the pyramidal tracts, leading to an excessive irritability of the cells of the anterior horns with the same result. He concludes from this and other cases that tabes in patients presenting evidences of syphilis does not differ in the clinical picture from those cases where no such evidence exists.

McCarthy.

225. DAS VERHALTEN DES RÜCKENMARKES BEI REFLECTORISCHER PUPILLENSTARRE (The Spinal Cord in Reflex Iridoplegia). G. Wolff (Archiv. f. Psychiatrie, 32, 1899, p. 57).

Wolff believes that not enough attention has been paid to the degenerations in the cervical cord in cases with the Argyll-Robertson pupil. He contributes the results of a number of autopsies with the microscopical findings in patients having this syndrome, mainly with tabes. He divides his observations into four series: 1. Pupils involved but reflexes normal. All of these, eight in number, showed a degeneration of the cervical cord in the posterior columns. In some there was also a degeneration in the lateral columns as well. 2. This group with pupils involved and decreased reflexes, seven cases, all showed changes in the cervical cord. 3. In one case with normal pupils and lost reflex knee phenomenon, the cervical cord was normal, but from the middle of the dorsal region down the degeneration of tabes was manifest. 4. In five patients showing normal pupils and normal or increased reflexes there were various findings. The degenerated areas were not constant, however, and were of different intensities.

JELIFFE.

226. DIE ERYTHROMELALGIE (Erythromelalgia). B. Sachs and Alfred Wiener (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, Nos. 3 and 4, p. 286).

The case of erythromelalgia reported by Sachs and Wiener is as follows: A man of middle age began to suffer severe pain, radiating upwards and downwards, in the left calf. Diffuse redness and swelling

of the foot appeared, and the symptoms were much exaggerated when the foot was pendent. Some months after this "red neuralgia" began; a small ulcer appeared on the dorsal surface of the foot, became deeper and wider, and dry gangrene of the second toe, and later of the other toes, of the foot developed. Amputation through the thigh was performed. The patient recovered from the operation and was free from pain. The portions of the anterior tibial and the popliteal arteries examined were found diseased, and the intima was most affected. The lumen in some parts was occluded. The changes in the nerves were proportionally slight. The authors believe that much more importance is to be attributed to arterial disease than to nerve degeneration in erythromelalgia, and that a condition similar to that of erythromelalgia without pain is not infrequently found in chronic cardiac and vascular disease. They report two cases in demonstration of this fact, and in conclusion they say that erythromelalgia occurs often as an independent symptom-group, although it is not an independent disease. In uncomplicated cases it is probably due to disease of the peripheral arteries. The obliterating endarteritis may be connected indirectly with central disease, but may be independent of such disturbance. In comparison with cardiac and vascular disease, erythromelalgia is probably more a disease of the arteries than of the nerves. SPILLER.

227. NORMAL AND PATHOLOGICAL HISTOLOGY OF THE NERVE CELL.
W. F. Robertson (Brain, 22, 1899, p. 203, Summer.)

Attention is here directed to this very excellent critical résumé of the recent literature bearing on the histology of the nerve cell. As the paper in question is a critical digest it cannot be abstracted. Particular attention is paid by the author to the histology; the pathology is given in bibliography. With this paper and its bibliography; with the monograph of Ewing's on "Studies in Ganglion Cells," and the "Bibliographical Contribution" of Jelliffe, both of which latter are published in the Archives of Neurology and Psycho-pathology, the present-day student cannot plead ignorance of the great field of ganglion cell pathology. JELLIFFE.

228. UEBER VOM VIERHÜGEL, VON DER BRÜCKE UND VOM KLEINHIRN ABSTEIGENDE BAHNEN (Concerning Descending Tracts from the Corpora Quadrigemina, Pons and Cerebellum). M. Probst (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, Nos. 3 and 4, p. 192).

Probst's paper is so full of detail that an abstract can hardly do justice to it. He finds experimentally that v. Monakow's bundle arises in the nucleus ruber and not in the thalamus or corpora quadrigemina, and it contains also ascending fibers. The descending fibers of the bundle could be traced into the sacral cord. The tract within the cord forms part of the crossed pyramidal tract, and because of this the area of degeneration in the lateral column is greater from a lesion in the spinal cord than from one in the pyramids or higher up. The normal fibers found within the crossed pyramidal tract in advanced cases of amyotrophic lateral sclerosis belong to v. Monakow's bundle. The tract has probably motor function, and yet division of the tract in animals does not cause paralysis. v. Monakow's bundle also receives fibers from the arched fibers (Bogenfasern) of the tegmentum of the pons.

The corpora-quadrigena-anterior-column tract (Vierhügel-Vorderstrangbahn) almost disappears in the lower cervical cord. It

gives fibers like v. Monakow's bundle to the anterior horns of the cord, and is probably motor in function.

The cerebral root of the fifth nerve descends lower than the exit of the fifth nerve, and can be traced in frontal sections so long as the vago-glossopharyngeus root fibers can be seen in their exit from the medulla oblongata. The fibers of the cerebral root have relations with the vago-glossopharyngeus nucleus, and the root also contains some ascending fibers.

Probst describes a new bundle which arises in the tegmentum of the pons and descends in the lateral column of the opposite side of the cord. Numerous motor tracts are found in the anterior, as well as in the lateral columns. The direct pyramidal tract in the dog and cat is usually unimportant, but may occasionally be well developed. The anterior column contains the corpora-quadrigenina-anterior-column tract (Vierhügel-Vorderstrangbahn), the fibers of the posterior longitudinal bundle arising in the deep nucleus of the posterior commissure and fibers from the lateral tegmentum of the pons. The bundle from Deiters' nucleus to the ventral peripheral zone (Rand-zone) contains ascending and descending fibers.

SPILLER.

CLINICAL NEUROLOGY.

229. ON THE RELATION OF THE NERVOUS SYSTEM TO DISEASE AND DISORDER OF THE VISCERA. Morison (Edinburgh Medical Journal, 1898, iv., 6, and 1899, v., 1, 2 and 3).

These, the Morison lectures for 1898, are practically the continuation of a course already reviewed in this journal, and are mainly devoted to giving the author's views as to what deductions should be drawn from anatomical facts, as well as from theoretical considerations. The subject is considered under the following heads: 1. Disorders of visceral innervation. 2. Disorders of visceral sensibility. 3. Disorders of visceral motion. The pathology of the visceral nerve ending is non-existent, or, at any rate, unknown, so the author proceeds to consider the finer nerve branches to the different viscera in their normal and pathological condition. As a type of an organ undergoing enormous changes in bulk he takes the uterus and studies its nerve supply in non-gravid and gravid conditions. By a comparison of sections through this organ, made at different periods, he finds that there is some increase in size of the nerves during pregnancy, though it is more apparent than real, as there is no increase in the number or size of the individual nerve fibers. There is, however, greater pliability and looseness in their texture. To account for the nerve supply to the hypertrophied uterine musculature, the author calls attention to the tortuosity of the uterine nerves, especially found in early pregnancy and post partum, the coiling being so marked as to suggest to him "the figurative parallel of paying out a coil of rope to an object, which is increasing its distance from a point of attachment." It is suggested that perhaps the onset of labor pains is due to the fact that the object has gotten to the end of its tether, and "the note is sounded for a return of the organ to more moderate dimensions."

In support of this idea the frequency of premature labor in hydramnios and twin pregnancy is adduced. He next passes to a consideration of the pathology of the ganglion cell and its connections, peripheral and central, but frankly states that the course of the visceral fibers in the central nervous system is too little known at present for us to be able to consider with any profit, lesions associated with "gen-

eral trophic degradation." Throughout the body there is "a functional unity, which might be more accurately termed a triunity, due to the consentaneous operation for the production of a common result of the peripheral organic cell—whether in brain or muscle—a central regulating cell, and its nerve channels, and the bond of unity between the two, the blood." Next follows a long discussion of the innervation of the heart and vessels, in which the author refers to the researches of himself and others, after which the production of visceral pain is taken up. Referring back to the uterus, he considers some further points in the anatomy of its nerve supply and concludes that the local causes of pain in the parturient organ are uterine and extrauterine, the extrauterine being such as are caused by pressure on surrounding structures, while the uterine are directly proportional to the degree of contraction, and are probably due to the squeezing of sensory nerve structures.

The pain of an ordinary muscle cramp is probably to be explained in the same way. Visceral pain may be absent, even in profound involvement of a viscus, where there is neither inflammation nor production of muscular spasm. Again it may be the peripheral expression of a central lesion.

One of the most interesting parts of the paper is that dealing with the subject of cardiac pain, especially the pain of angina pectoris.

The nerves of the heart are in intimate relation with the cardiac blood vessels and must undoubtedly be affected by changes in their caliber and by alterations of their walls. There again squeezing of the sensory structures and stretching of the nerves—in enlargement of the heart—most probably plays a rôle. Cardiac pain, however, may be of extracardiac origin, as witness the cardiac crises occurring occasionally in tabes, not to speak of the merely reflected pain not uncommon in disturbances of the abdominal viscera. As determining the central causation of cardiac pain little is known, but all the evidence we have is in favor of the spinal, rather than the cranial, nerve supply of the heart as "the chief territory of objective pain."

The pain of meningitis and of some headaches, the author thinks, may arise in the nerves of the membranes of the brain, but on account of the brain being the "seat of perception and the focus of the sensory channels of the body" disease implicating its centers and nerves may cause pain in a manner impossible in any other viscus. Passing on to disorders of visceral motility, the author again takes up the heart, and enters into a discussion of the causes of its motility, which space does not permit us to follow. He next proceeds to show how visceral disorder may underlie somatic motor phenomena as well as sensory phenomena. Last, turning to the brain, he considers it in its receptive, retentive (or connective), and executive functions, and reviewing the character of the nerve cells in different regions of the cortex, gives the reasons for thinking that these functions are there represented in different regions to different degrees. The above gives but a bare outline of these lectures. Those specially interested are referred to the original. The article is well illustrated. ALLEN.

230. ZUR DERMATOMYOSITIS (Contribution to Dermatomyositis). H. Oppenheim (Berliner klin. Wochens., 37, 1899, p. 805).

Two cases of dermatomyositis, or dermatomucosomyositis, are reported. The first case was a boy of eight years, who exhibited an erythematous condition of the skin of the hands, arms and face; edema more especially in the upper facial areas; intense pain on motion; sensitiveness to pressure over the affected muscles, and increased tempera-

ture. The lad assumed a stiff, rigid position to protect the affected musculature. Paresis, with atrophy of some muscles, was noted, but electric examination produced such intense pain that it had to be omitted. There was an abnormal condition of tension in the affected muscles, and on palpation these muscles felt hard and sinewy. The mucous membranes were reddened, and the patient complained of a burning sensation in the mouth and throat, made worse by attempts at swallowing. Swelling of the cubital and cervical glands Oppenheim considers secondary. Myositis ossificans is excluded on account of the absence of areas of ossification and the involvement of the mucous membranes. Trichinosis is excluded by the absence of gastro-intestinal involvement. The second case was a fifty-year-old gymnasium instructor, who immediately after a "Kneipp cure" developed a stomatitis, angina, marked edema of skin, and intense pain throughout the muscular system. Ulceration of the affected skin areas occurred, attacks of fever and bronchitis developed, and the patient showed signs of convalescence, but a rapid pulse and other symptoms of cardiac failure developed, from which he finally died. Whether the heart disease was due to the general muscular involvement or was a result of exhaustion, intoxication and increased temperature, Oppenheim was unable to decide. Microscopic examination of some of the diseased muscle of the first case showed a cellular infiltration of the muscle tissue. The perimysium was thickened and less marked changes were present in the muscle fibrils. These latter were for the greater part atrophied and at times completely destroyed.

McCARTHY.

231. UERER DIE AUTOCHTHONE HIRNSINUSTHROMBOSE (Concerning the Autochthonous Thrombosis of the Cerebral Sinuses). G. v. Voss (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, Nos. 3 and 4, p. 297).

Sinus thrombosis is not easily diagnosticated. Voss reports nine cases, and in eight of these the correct diagnosis was not made. The symptom-complex of sinus thrombosis resembles most closely that of acute leptomenigitis. Both diseases usually begin acutely, and headache is the most prominent symptom in both; disturbance of consciousness increasing quickly or gradually to coma; single, and later general, twitchings, tonic and clonic convulsions, paralyses, vomiting, vertigo, disturbances of the pupil, are common symptoms of both affections. Abnormalities of pulse and temperature, rigidity of the neck, pronounced hyperesthesia of the skin and muscles, and disturbances of the ocular muscles, usually occur in meningitis. Disturbance of temperature rarely occurs in sinus thrombosis, the pulse usually remains normal, rigidity of the neck is exceptional, and general hyperesthesia in muscles and skin has not been observed. Local signs of congestion (fullness of the veins of the face, nasal hemorrhage) are sometimes pronounced in thrombosis and are always absent in meningitis. Cachexia of long duration, chlorosis and anemia suggest thrombosis; whereas the presence of tuberculosis elsewhere in the body suggests meningitis. The differential diagnostic value of choked disk is uncertain. Localized disturbance of sensation (pain, hyperesthesia, anesthesia) occurs in thrombosis and not in meningitis.

The diagnosis between sinus thrombosis and cerebral tumor is also difficult. Slow development of the symptoms occurs in tumor; rapid development usually in sinus thrombosis. Distinct focal symptoms, disturbance of ocular muscles, nystagmus, paralysis, slowing of the pulse, tumor in other regions, would favor more the diagnosis of cere-

bral tumor; intense cachexia of long duration, anemia and chlorosis would favor thrombosis.

Sinus thrombosis resembles clinically other disturbances in the vascular system, encephalitis, cerebral abscess, etc. Obstinate and violent headache in young anemic or chlorotic women should arouse a suspicion of sinus thrombosis. Changes in the eye ground resulting from congestion of the central vein of the retina or the cavernous sinus make the diagnosis of sinus thrombosis easier. Cachexia, anemia or chlorosis of long duration with suddenly developed headache and other cerebral symptoms, should arouse a suspicion of sinus thrombosis. Local congestion, limited disturbance of sensation, anomalies of movement (twitchings, convulsions, chorea) without changes in the temperature or pulse, render the diagnosis of sinus thrombosis probable.

SPILLER.

- 232 UEBER AFFECTIONEN DER CAUDA EQUINA (Concerning Affections of the Cauda Equina). W. v. Bechterew (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, Nos. 3 and 4, p. 222).

Cases of disease of the cauda equina with necropsy are not very numerous. v. Bechterew reports a case in which a tumor developed in the left testicle, and was not removed until metastasis had occurred in the retroperitoneal region. The metastatic tumor penetrated the vertebral column, and caused pain by compression of the nerves. The vertebral column, already weakened by the ingrowth of the tumor, fractured during chloroform narcosis, and compressed the cauda equina in such a way that complete paralysis of the lower limbs was observed immediately after recovery from the narcosis. The symptoms in the case were anesthesia of the buttocks, the posterior part of the thigh, leg and foot, of the anterior surface of the leg, of the perineum, anus and penis; hypesthesia on the anterior surface of the thigh; paralysis of the gluteal muscles, of the muscles of the legs and flexors of the lower limbs; paresis of the extensors and adductors of both thighs; paralysis of the detrusor muscle and rectum; hyperesthesia of the scar in the scrotum and of the lower part of the abdomen; occasionally convulsive movements in the abdominal muscles. Paralysis of the sphincters was not very evident, but on the contrary paralysis of the detrusor vesicæ muscle existed. Cases of disease of the cauda equina with so extensive paralysis of sensation and motility, and with muscular atrophy, with relative integrity of the sphincters, have not been published, according to v. Bechterew. The fracture of the weakened vertebrae during narcosis was of much importance.

SPILLER.

THERAPY.

233. THE PHYSIOLOGICAL ACTION OF THE ALKALOIDS DERIVED FROM ANHALONIUM LEWINII. W. E. Dixon (Journal of Physiology, 25, 1899, p. 69).

This drug, known for a long time to the Mexican, was first brought into prominence in 1888 by Lewin, for whom it was named. It first attracted attention by reason of its vision-producing properties, the which are attested to by numerous observers. Dixon here undertakes a careful study of the four alkaloids isolated by Heffter in 1896, anhalonnine, anhalonidine, lophophorine and mezcaline. These have been isolated and show close chemical affinities; anhalonnine and anhalonidine are isomeric and the others are closely related. The action of the drug is slightly complex on account of the four alkaloids. There is an increase in salivary secretions, vomiting and diarrhea are

present from large, but not from small, doses, the blood is not affected, the heart is affected, probably directly in its muscular substance, it is slowed and the force of the beat is increased in a manner somewhat analogous to the action of digitalis. Respiration is not affected in small doses, but in toxic amounts it becomes quicker and shallower; death usually resulting from respiratory centric paralysis. The effects on the nervous system were described early by Weir Mitchell. In mammals the effects are characteristic. Cats purr and prefer the dark, are soporific and stare "fixedly" with widely dilated pupils. The deeper reflexes are all exaggerated, but cutaneous sensations are blunted to the point almost of anesthesia. Ataxia with swaying is prominent in the gait. It would seem there is a preliminary stage of excitement in the action of the drug on the cerebral centers; this is usually marked by a sense of exhilaration, without much merriment, talkativeness is present to a marked extent, and the patients feel light-headed. This stage is followed by one of intoxication, in which the tendency to lie down is manifest, though there is little or no inclination to sleep. The pupils are widely dilated and sluggish to light, inco-ordination in gait resembling alcoholism, is apparent and there is a well-marked generalized tremor. Muscular twitchings are not infrequent. Dual-existence sensations similar to those experienced in cannabis indica are not infrequent; the visual hallucinations are the most interesting and constant cerebral manifestations.

They seem at first to resemble the first optical sensations of migraine and then finally develop into other fantastic, bizarre and interesting shapes, though rarely of external objects. Coloring is intensified and often beautifully blended.

Some slight variation in the action of the alkaloids is apparent: Anhalonnine and anhalonidine are quite similar in their action, but lophophorine, while similar in action, is double the strength, especially in its action on the cord. Mezcaline acts apparently mostly on the visual centers. It is patent that "mescal" acts differently from any other known substance. It most closely resembles cannabis indica, but does not produce the feelings of merriment common to that drug. Its effects on the brain and cord are similar to those of strychnine, while its action on the heart suggests digitalis. Therapeutically, as a general stimulant to the nervous system it seems indicated and especially so in melancholia, where its action on the kinesthetic sensations render it applicable.

JELLIFFE.

234. THYMUS EXTRACT IN EXOPHTHALMIC GOITER, W. Rushton Parker (British Medical Journal, No. 1984, p. 12, Jan. 7, 1899).

The author reports four cases.

Case I. A well-marked case of three years' standing in a woman of twenty-eight. In six months she took 2,000 five-grain thymus tabloids, mostly ten to twelve daily, and ate a large number of lightly-cooked lambs' thymus glands, with no perceptible effect, although the patient "somehow professed to feel better at the end of the treatment than at the beginning."

The author incidentally remarks that iron, digitalis, bromide, beladonna, arsenic and blistering were equally ineffective.

Case II. A woman of thirty-five, of a goitrous stock in a goitrous valley, presented the typical symptoms of Graves' disease, except those relating to the eyes. During eighteen months she took 5,000 five-grain thymus tablets, usually twelve daily, and ate many scores of lambs' thymus glands. She improved somewhat as regards general symptoms, but as she had notably improved during the two preceding years, the effect

of the thymus was problematic. Digitalis, strophanthus and blisters were persisted in for some weeks with no effect.

Case III. A woman of forty-five; disease of about a year's duration. She took 400 five-grain tablets in three months, besides many lambs' thymus glands. During this time there was some improvement, but the pulse remained at 160 to 200, but in the following six months, during only one of which thymus was taken, she continued to improve at at least an equally rapid rate. "This patient, originally severely and dangerously ill, became practically quite well; but the improvement began long before the administration of thymus, and seemed to continue independently of it; moreover, thymus was taken in comparatively small doses of four to six tabloids daily." Digitalis at first seemed beneficial, but subsequently did more harm than good.

Case IV. A woman of forty-nine was first given thyroid tablets for six weeks, but they distinctly aggravated the symptoms. During the next year she took 700 five-grain tablets of thymus with some improvement, but during three consecutive months of this period she took no medicine and her condition remained as good as during the administration of the remedy. Nevertheless, the patient was found a year and three-quarters subsequently perfectly free from all discoverable symptoms except a pulse of ninety-six, and a very slightly enlarged thyroid, having throughout that period treasured up her last stock of thymus tablets, convinced that they had cured her of her troublesome symptoms and might therefore be needed at some future time.

PATRICK.

235. UEBER DIE BEHANDLUNG DER SYPHILIS IM ALLGEMEINEN UND ÜBER DIEJENIGE DER TABES POSTSYPHILITICA IM BESONDEREN (Concerning the Treatment of Syphilis in General and especially of the Post-syphilitic Tabes). S. Tschiriew (Monatsschrift für Psychiatrie und Neurologie, Vol. 5, No. 6, 1899, p. 440).

Tschiriew has had an experience of more than seventeen years in military hospitals, and has found that the most common affections among army officers are the postsyphilitic. Syphilis, he says, is a constitutional disease and never completely curable, although post-syphilitic affections may be warded off if specific treatment is repeated at least once every three years during the ten or fifteen years following the infection. All excesses must be avoided. He disapproves of the simultaneous administration of mercury and the iodides. The latter hasten the elimination of the mercury, and thereby lessen its effectiveness in the treatment of syphilis, and increase the danger of stomatitis. Mercurial inunctions are of unquestionable value; no serious objection can be made to the mercurial ointment, although the mercurial soap employed by the physicians of Aix-la-Chapelle is preferable. Baths increase the circulation of the mercury in the system, and the elimination through the kidneys and sweat glands, and thereby lessen the elimination through the mouth. A bath of 35 degrees C. lasting from twenty to thirty minutes should be given daily, and inunctions should be made a half to one hour after the bath. Inunctions should be given on six successive days, and on the seventh day a bath should be ordered. This treatment should be employed for five or six weeks, and during this time the mouth should be washed out seven or eight times daily with a solution of chlorate of potash, especially after eating, and the teeth and even the tongue should be brushed. The functions of the stomach and intestines must be carefully regulated, and the use of tobacco restricted. Tschiriew employs five to six grams of mercurial soap or mercurial ointment daily. Pills and subcutaneous injections of mercury are not to be recommended. The inunctions are

discontinued after they have been employed for five or six weeks, the baths are then given three times weekly, and the iodides are employed in the dose of 0.5 to 4.0 grams three times daily. Water from a sulphur spring may be advantageously administered during the "cure."

Tschiriew's treatment of tabes consists of douches, electrization of the spinal cord, medulla oblongata and urogenital apparatus, applications of the Pacquelin cautery along the spine, etc. SPILLER.

236. THE PHYSIOLOGICAL EFFECTS OF EXTRACTS OF THE PITUITARY BODY. E. A. Schäfer and Swale Vincent (*Journal of Physiology* 25, 1899, p. 87.)

The authors believe that there are specific, active substances in the infundibular part of the pituitary body and conclude from a series of experiments that:

(1) Extracts of the pituitary body, when intravenously injected, have a marked effect upon the blood pressure, producing, according to the nature of the extract, either a marked rise or a marked fall. The pituitary body contains, therefore, two active substances, one *pressor* and the other *depressor*. Of these the pressor substance is soluble in salt solution and insoluble in absolute alcohol and ether; the depressor substance is soluble in salt solution, in absolute alcohol, and in ether. The active substances are not destroyed by boiling and are dialysable.

(2) The pressor substance produces its action both upon the heart and upon the peripheral arteries. (The action of the pressor substance is a prolonged one, and during the period of its action a second dose is inactive, or nearly so.) The action of the depressor substance is evanescent and can be repeated at short intervals.

(3) The pressor effect of the extract may be accompanied by cardiac slowing. This is probably in large part incidental to the contraction of arterioles and rise of aortic pressure, but is in part due to direct action upon the peripheral mechanism.

(4) The active substances are contained only in the infundibular, not in the hypophysial, part of the pituitary body.

(5) Subcutaneous injection of the extracts in small mammals causes paralytic symptoms similar to those obtained by injecting suprarenal extracts.

(6) The characteristic effects produced by extracts of the infundibular body are probably not due to the gray nervous matter of which this is largely composed. JELLIFFE.

237. MYXEDEMA TREATED WITH "COLLOID" MATERIAL. Robert J. M. Buchanan (*British Medical Journal*, No. 2007; June 17th, 1899, p. 1460).

A man of fifty-four years who had presented the symptoms of myxedema for a period of two years or more was treated with Oppenheimer's "palatinoids" of thyroid "colloid."

Treatment was begun with one palatinoid at night which was gradually increased to four in twenty-four hours, and then reduced to two a day, and later to one a day. There were no symptoms of thyroidism, and at the end of five months the patient was considered to be entirely normal in every respect. He continues to take one or two of the palatinoids daily.

For a full account of the chemistry and preparation see *British Medical Journal* of March 21st, 1896; January 23d, 1897, and February 17th, 1897; *Journ. Physiol.*, Vol. XX, p. 474. PATRICK.

238. LE TRAITEMENT DE LA MORPHINOMANIE PAR LA SUPPRESSION BRUSQUE (Treatment of Morphinomania by Abrupt Suppression). A. Lutared and B. Deering (Revue de Psychiatrie, 1899, p. 52).

In this critical digest of more recently expressed opinions relative to the treatment of this drug habit the authors hold with many observers, more particularly Germans, that the sudden withdrawal of morphine does no marked harm to the patient. French writers especially have taught that such withdrawal was usually attended with grave accidents. The authors believe that in most cases, unattended by fatal organic disease with pain, the drug can be entirely suspended after from four to eight days, and they further claim that such a means is the only rational one to follow in the treatment of this condition. JELLIFFE.

239. SUR LE TRAITEMENT AU LIT DES MALADES PSYCHIQUES AGITÉS (On the Bed Treatment of Agitated Mental Conditions). J. Kostetsky (Questions de médecine neuro-psychique, 1, 1899, p. 31), Revue Neurologique).

The author presents a good summary of the literature and reports on his experiences with 75 cases of disturbed patients among women under bed treatment. He calls attention to the fact that some patients accustom themselves early to the method and remain in bed without much supervision, while others are broken in with difficulty and require constant watching for a time. On admission a certain amount of depression is noted by reason of seeing so many patients in bed, but this does not seem to materially affect their mental condition.

Patients with systematized delirious states seem to yield quite readily. Idiots and demented are very hard to manage in acute outbreaks. In secondary dementias, with agitation, the bed acts beneficially, especially with reference to hygiene; it conduces to cleanliness and quiet. In acute maniacal outbursts, the attack does not seem shortened. In melancholia, associated with anemia, excellent results are reported. Also in acute confusional states. In "folie morale" the author has had good results only with the co-operation of the patient.

Kostetsky seems to think that both acute and chronic cases are benefited by the bed treatment. It helps the digestion; sleep seems to improve, the patient gains in weight. Indolence is to be avoided. In all such cases, if it is practicable, lying in bed in the open air is recommended. He especially commends the method for the treatment of patients recently admitted. JELLIFFE.

240. LE TRIBOMURE DE SALOL; SA VALEUR COMME HYPNOTIQUE CHEZ LEZ ALIÉNÉS (Tribromide of Salol as a Hypnotic for the Insane). Viallon (Annales médico-psychologiques, 9, 1899, April).

Of the numerous new hypnotics more recently proposed and experimented upon tribromide of salol seems to offer some promise.

The author has employed it in some thirty-five cases of miscellaneous forms of insanity. Its greatest efficiency would seem to be manifest in chronic mania with agitation, and in dementias with temporary excitement. In other types its action would seem to be negative. Among its disadvantages, high price, irregularity of action and its limited range of application should be borne in mind. JELLIFFE.

PSYCHIATRY.

241. SULLA CLASSIFICAZIONE DEGLI STATI PSICOPATICI (Classification of Psychopathic States). G. Angiolella (Il Manicomio Moderno, 15, 1899, p. 1).

The author in a critical review of some seventy-two pages discusses

the various classifications of ancient and modern writers and contributes one of his own, as follows:

- I. Psychological Anomalies.
 1. Total arrest of psychical development.
 - (a) By defective organic evolution.—Idiocy, imbecility.
 - (b) By defective metabolism.—Myxedema, idiocy, cretinism.
 2. Partial arrest of psychical development.
 - (a) Partial idiocy.
 3. Deviation of psychical development.
 - (a) Affecting intellect.—Superior degenerates.
 - (b) Affecting volition.—Impulsive characters, passionate delinquents, suicides (impulsive and melancholic).
 - (c) Affecting sentiments.—Mattoids, primary paranoia, querulomania, moral insanity, sexual pervers.
 - (d) Constitutional anomalies with intercurrent morbid phenomena.—Epilepsy, hysteria, neurasthenia (fixed ideas), hypochondriasis.
- II. Mental Diseases in Strict Sense.
 - A. Degenerative Diseases, Degenerative Psychoses.
 1. Psychoses dependent upon organic development.—Hebephrenia (catatonia), insanity of menopause, senile psychoses (mania, delirium, dementia).
 2. Chronic psychopathic states.—Late systematized paranoia, periodic insanity, recurrent insanity, circular insanity.
 3. Acute psychopathic states.—Acute hallucinatory delirium, degenerative mania, delirious lipomania.
 - B. Diseases developing after simple cerebral weakness.
 1. Due to minute curable lesions of nerve cells.
 - (a) States of ideoaffective excitement.—Mania.
 - (b) States of sensory excitation.—Sensory delirium.
 - (c) States of psychical pain.—Lipomania.
 - (d) States of depression.—Mental confusion (febrile delirium), acute paranoia, stuporous and somnambulistic states.
 - (e) States of psychical—transitory neurasthenia, consequent on acute psychoses.
 - (f) States due to disturbed cerebral nutrition by lesions in other organs.—Reflex insanities, acquired hysteroid states.
 2. Due to grave and permanent lesions of nerve cells.—General paresis, post-apoplectic dementia, dementia of cerebral tumors, dementia of multiple sclerosis, dementia of localized meningitis, epileptic and traumatic insanity.
- III. Psychological Affections Consecutive to Psychopathies.—Secondary Paranoia, Consecutive Dementia, Post-paranoid Dementia.

JELLIFFE.

242. LES AUTOINTOXICATIONS DANS LEURS RAPPORTS AVEC LES DÉLIRES (Autointoxication in Its Relation to Delirium). Régis (Archives cliniques de Bordeaux, 1898, Nos. 9 and 10).

This is an abstract of a work which obtained the Aubanel prize of the Paris Medico-Psychological Society for 1898.

The author takes up the subject of the relations existing between autointoxications and mental disorders, considering them under the following heads:

- A.—Mental disturbances due to autointoxication.
- B.—Autointoxication occurring in mental disorders.

Stating that many volumes would have to be written if the insanities connected with all varieties of autointoxication were to be

described, and calling attention to the fact that psychical troubles arising from autointoxication of whatever kind present great general analogies, the author announces his intention of confining himself first to the detailed description of the mental disturbances connected with the principal autointoxications (gastro-intestinal, hepatic and renal), and, second, to showing that insanities arising from autointoxications of other sorts are practically identical in character with these. As a type he takes the mental disturbances arising in the course of gastro-intestinal autointoxications, describes first the nervous and elementary psychical troubles and then the true psychoses, considering separately those in acute and those in chronic gastro-intestinal affections.

Many of these morbid mental manifestations have undoubtedly a close relationship to neurasthenia, and arthritic heredity can be traced in many cases. Mixed forms also occur. The particular point made, however, is that by being able to recognize gastro-intestinal autointoxication as the chief underlying cause, and by applying treatment addressed to this condition, most of such cases can be improved—many cured. The author relates the histories of a number of illustrative cases, drawn both from his own and from the experience of others.

ALLEN.

243. THE LEGAL VERSUS THE SCIENTIFIC TEST OF INSANITY IN CRIMINAL CASES. Carlos F. MacDonald (American Journal of Insanity, 56, 1899, p. 20).

The plea of insanity as an excuse for crime is daily becoming more prominent, and this discussion by Dr. MacDonald is a very timely and valuable one. From a medical point of view the author holds that there is much need of the reconstruction of present tests for determining responsibility. Insanity and irresponsibility are convertible terms, and, according to our author, whenever the existence of insanity is clearly established the question of responsibility is practically determined. "Hence this question of responsibility for criminal acts is, strictly speaking, a medical one, and it can only be determined, especially in complex and obscure cases, by those who are practically familiar with the symptoms of mental derangement." The law holds that the question of responsibility is not to be settled by the mere *existence* of insanity, but by the *degree* or *extent* of its existence, and the legal test attempts to draw an arbitrary line between insanity and irresponsibility which is at variance with nature as studied by medical science. The law does not recognize a form of insanity in which the capacity of *distinguishing* right from wrong exists without the *power of choosing* between them. Which means the totally wrong position, known to most alienists, "that an individual who possesses a knowledge of right and wrong must necessarily possess the power of choosing the right and resisting the wrong with reference to any particular course of action, and that such a man is legally sane and responsible, no matter how far his mind may be unhinged in other directions." This position, Dr. MacDonald points out, is one taken as early as 1843, and it would seem that legal science, in this line, had not progressed beyond that time.

The real question of fact for the jury to determine in cases of alleged insanity in criminal trials, as pointed out by the writer, would seem to be the following:

1. Did the defendant at the time of the alleged crime have sufficient mental capacity to *rationaly* appreciate the nature and consequence of the act he was committing, and if so, had he sufficient power of will to enable him to choose between doing or not doing it?

2. If he had lost the power of choosing, with reference to the par-

ticular act, was the loss due to disease, and not to "heat of passion," intoxication or other self-induced temporary mental disturbance?

JELLIFFE.

244. OLFACTORY HALLUCINATIONS IN THE INSANE. F. St. John Bullen (Journal of Mental Science, 45, 1899, p. 513).

From a series of 95 cases in whom olfactory hallucinations were presumed to be present, collected from various asylums, the author presents some interesting observations. In a few cases the distinction between illusions and hallucinations were not possible. Such hallucinations are most commonly found among the chronic delusional psychoses, especially connected with persecutory ideas. The author does not believe that there is any direct relationship between the reproductive system and the sense of smell, but that there is some underlying connection cannot be entirely excluded. Inasmuch as smell occupies such a minor role in the relations of society, it seems that it is tardily affected in the breaking down of the insane, but if found it would seem to indicate some fundamental and grave defect. It can only be looked for when some close and often excited association is aroused, such as that of taste, or when some general implication of the senses, of a fundamental kind, exists. Much evidence must be required to show that an hallucination of the olfactory sense when occurring singly, is directly related to a morbid condition of the reproductive system, but when a connection is affirmed in the presence of several hallucinations—*i. e.*, of a general sensory disturbance—there must be a danger of overlooking other factors in the case. It is not possible, however, to write except as to the possibilities in this subject, for the authors who have laid stress upon an olfactory-sexual nexus may have had the very instances in support of their views that the present writer has failed to secure. Such an association would show much more distinctly when met with in early cases of insanity and among the more educated classes, whose sensations are better observed, and in whom the function of smell has been enlarged by more varied and numerous experiences than have fallen to the lot of the pauper inmates of asylums.

JELLIFFE.

245. CAUSES OF THE INCREASE OF SUICIDE. W. W. Ireland (Journal of Mental Science, 45, 1899, p. 451).

Ireland in a careful review of the general subject of suicide gives some interesting facts and ideas worthy of epitomizing. Among other things he says: "Most of the causes which impel men to seek their own death are the same in the sane and insane. People get weary of life and seek death because they are unhappy. Their misery overpowers the natural love of life. Self destruction in the sane naturally takes place amongst persons who feel hopelessly ruined or disgraced, or subject to some pain or overwhelming affliction, from which they are unable to escape. Though a few men may destroy themselves in cold blood, it may safely be inferred that before being impelled to an action so repugnant to nature, most men are in a very troubled or distracted frame of mind. For some time their minds have been unhinged; they have lost their appetite for food, or have been sleepless for many nights. The unquiet mind has acted upon the body, and the disturbed bodily functions have reacted upon the mind." A practice so widely spread as suicide undoubtedly has many causes, yet the mental factors are believed to be the most potent. Among these the religious feelings are interesting. Suicide seems most common among those holding materialistic or fatalistic doctrines, and it is to the supposed increase in materialism that the alleged increase in suicides in Europe and North America is due. And where agnosticism most abounds, as in large cities, suicides are most common. [Other factors abound in such places also.]

JELLIFFE.

Book Reviews.

DIAGNOSTIC DES MALADIES DE LA MOELLE, SIÈGE DES LÉSIONS. Par le Dr. Grasset, Professeur de Clinique à l'Université de Montpellier, Associé National de l'Académie de Médecine, Lauréat de l'Institut. Paris: J. B. Baillière et Fils, 1899.

We have already had occasion to praise for their timeliness and conciseness those numbers of the excellent little series entitled "Les actualités médicales," which have previously appeared, and this, the fourteenth, is in no way inferior to its predecessors. Its object is to assist in exactly locating the lesion in cases of spinal disease, and to this end its 92 pages are divided into two sections. The first of these correlates each group of symptoms with its corresponding horn or column lesion or lesions, while in the second the means are discussed by which it is possible to determine at what points in its length the cord is affected.

The little brochure fulfills admirably the end of presenting much information in compact form and deserves hearty commendation.

VOGEL.

BOOKS RECEIVED.

"Index Catalogue of the Library of the Surgeon-General's Office, United States Army." Vol. IV.

"Treatment of Pelvic Inflammation through the Vagina," by Wm. R. Pryor, M.D. W. B. Saunders, Phila.

"Text-Book of Diseases of the Nose and Throat," by D. Braden Kyle, M.D. W. B. Saunders, Phila.

"The Hygiene of Transmissible Diseases," by A. C. Abbott, M.D. W. B. Saunders, Phila.

"American Pocket Medical Dictionary," by W. A. Newman Dorland. W. B. Saunders, Phila.

"Progressive Medicine." Vol. 3. Sept., 1899. Lea Bros & Co., Philadelphia, Pa.

"International Text-Book of Surgery," by J. C. Warren, M.D., and A. P. Gould, M.D. W. B. Saunders, Phila.

"Raynaud's Disease," by T. K. Monro, M.D. Jas. Maclehose & Son, Glasgow.

"The Cerebro-Spinal Fluid," by St. Clair Thomson, M.D. Cassell & Co., London, Eng.

"Diagnostic des maladies de la moelle siège des lésions," by Dr. Grasset. J. B. Baillière, Paris.

"Les troubles mentaux de l'enfance," by Dr. M. Manheimer, Paris.

"Die Nervösen Krankheitserscheinungen der Lepra," by Dr. Max Laehr. Geo. Reimer, Berlin.

"Il nervosismo di questa fine di secola." Prof. Leonardo Bianchi.

"Su le idee fisse." Prof. Leonardo Bianchi.

"Contributo alla diagnosi e alla cura delle astralgie isteriche." Prof. Leonardo Bianchi.

THE
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OF
Nervous and Mental Disease

Original Articles.

THE COMMON FORMS OF MENINGITIS, AND THEIR RECOGNITION, WITH SPECIAL REFERENCE TO
SEROUS MENINGITIS.

By CHARLES L. DANA, M.D., of New York.

I do not know of a subject that is much more perplexing to a student than that of the different types of meningitis. The classification, never very simple, has undergone constant changes during the last half century, and such changes have been numerous even within the last years. The introduction of various kinds of serous meningitis and meningo-encephalitis, as well as of non-purulent encephalitis, and the peculiar manifestations of cerebral edema, have served to increase the complexity of the subject. The inflammations of the dura mater have also caused much discussion and given rise to much variety in definition and classification.

In one of the earliest and most conscientiously-made classifications of meningitis, that by Huguenin, published in Ziemssen's "Cyclopedia," and written nearly twenty-five years ago, fourteen different forms of cerebral meningitis were described. Grasset's work, which was published about the same time, and is a very masterly treatise of its kind, illustrates the different qualities of the French and German mind, for he describes only acute, chronic and tuberculous meningitis. The progress since Huguenin's time has been shown, on the one hand, by a gradual simplification of the classification, and on the other, by clinical and pathological studies which have added to the actual richness of the field, and obliged us to put two or three

new names in the list. Thus, Gowers, Oppenheim and Mills, although they drop out many of the terms used by Huguenin, still give us from ten to twelve types of meningitis.

There are classifications of diseases which must be elaborate, because we wish them complete, but in many cases this completeness is not necessary in practical medicine and destroys in the student the sense of proportion between what is vital and what is not important. At present we can find in literature: *A.* Pachymeningitis, external: (1) acute, and (2) chronic; internal: (3) hemorrhagic, (4) syphilitic, (5) purulent, and (6) serous. *B.* Leptomeningitis: (7) acute, and (8) chronic simple; (9) acute, and (10) chronic syphilitic; (11) acute and (12) chronic epidemic; (13) tubercular, (14) posterior basic. *C.* Serous meningitis: (15) acute ventricular, (16) acute general, (17) traumatic, (18) benign, (19) malignant, (20) chronic. This makes 20 different forms, without using the term "meningismus," or including acute cerebral edema or acute and chronic hydrocephalus.

The question arises, How many of the forms have actual clinical existence or importance? Without any special discussion we can, I think, agree that external pachymeningitis, (1) acute and (2) chronic, is only a surgical complication, and is probably never an independent malady. We can also drop purulent internal pachymeningitis (5), while serous internal pachymeningitis (6) is only a pathological curiosity. Acute syphilitic meningitis (9) is certainly most rare, if it ever exists, at least, using the word *acute* in the same way we apply it to other forms. Posterior basic meningitis (14) is probably a syphilitic or tubercular disease or a serous meningitis. Serous meningitis has three forms, as I shall try to show later. The malignant (18) and chronic (19) forms have a doubtful standing. We thus reduce the 19 types of meningitis to 11, or, dropping all temporal distinctions, to 9.

The forms of meningitis as seen in ordinary practice are shown by an analysis of 137 cases which I have collected from the records of Bellevue Hospital, the majority occurring in my service in the second and fourth medical divisions of the hospital during the past fifteen years. Among these, there were 46 with autopsical records.

FORMS OF MENINGITIS IN 137 CASES AT BELLEVUE HOSPITAL.

External pachymeningitis (from mastoid disease).....	2
Internal pachymeningitis (hemorrhagic and syphilitic)...	12
Fibrinopurulent leptomeningitis.....	52
Cerebrospinal leptomeningitis.....	15
Tuberculous leptomeningitis.....	14
Serous meningitis { traumatic	
alcoholic	15
infectious	
Chronic leptomeningitis.....	3
Unclassified forms.....	19
Spinal meningitis.....	5
Total	137

CASES WITH AUTOPSY.

Cerebrospinal leptomeningitis.....	5
Purulent cerebral leptomeningitis.....	18
Tuberculous leptomeningitis	7
Serous leptomeningitis.....	7
Pachymeningitis interna.....	9
Total	46

Leaving out the spinal cases there are, therefore, only five kinds of meningitis recognized in ordinary hospital practice. It will be seen also that the purulent, tubercular, cerebrospinal and serous are the principal forms, while pachymeningitis is practically only a chronic syphilitic manifestation. These hospital figures, I am sure, coincide in the main with the ordinary clinical experience of all of us, but I put them forth here to emphasize the importance of these forms and the relative insignificance of the other types.

Recognition of the Different Forms. Cerebral and Spinal Meningitis. In speaking of *meningitis* of the pia-arachnoid it is often unnecessary nowadays to use the term "cerebral" or "spinal." Practically all cases of acute cerebral meningitis are cases of cerebrospinal meningitis; on the other hand, acute spinal meningitis is a very rare disease. Indeed I have never seen a primary case and can find no record of one. We can almost, therefore, abandon the distinction between cerebral and spinal meningitis in ordinary clinical work. I have rarely failed

to find pus in the spinal canal if it is abundant on the cerebral membranes. Tubercles are also often found in the spinal canal if there is cerebral tubercular meningitis.

Pachymeningitis is a disorder which has received an extraordinarily rich vocabulary of qualifying terms. The distinction into external and internal types is a very academic one. Pus on the dura, or extradural abscess, is a more correct term for acute suppurations involving the outer surface of the dura. Here, mastoid inflammations and other forms of osteitis are really the disease, while the meningitis is simply an annex to it.

A non-surgical pachymeningitis is mostly an *internal* pachymeningitis, although naturally the whole membrane is more or less deeply affected. This form of inflammation is commonly syphilitic, and next to that, hemorrhagic. The hemorrhagic form is associated with disease of the blood and blood-vessels and is found practically only in scorbutic children, in insanity and alcoholism.

Leptomeningitis. The inflammations of the pia mater have also led to a luxuriant growth of types and names. There are very few writers who have not given a special classification of their own, so that meningitis vies with insanity in the disheartening multiplicity of its forms. Yet, practically, we know that at the bedside nearly all the forms of meningitis have pretty much the same symptoms, differing mainly in the rapidity or intensity of the process. The common forms, as I have shown, are the epidemic (cerebrospinal), fibrinopurulent and tuberculous.

Epidemic cerebrospinal meningitis can usually be readily recognized. The history of an epidemic, absence of trauma or previous infectious fever, the skin symptoms, the greater involvement of the base of the brain and cranial nerves suggest the nature of the trouble. The employment of lumbar puncture enables us to settle the diagnosis, if we find the diplococcus intracellularis meningitidis or micrococcus lanceolatus. There seem to be some epidemics due to the latter organism, and there is no way of distinguishing the two forms except by culture. In two cases in which the patients were tapped and examined under my observation the diplococcus was found.

Simple fibrinopurulent meningitis may be due to almost any pyogenic organism; usually it is a streptococcus infection. It is not rare, however, to find the pneumococcus. We do not know the distinctive clinical features of pneumococcus meningitis, though it seems to be a very fatal form.

Tuberculous meningitis. There is a form of inflammation which, it is quite agreed, has distinct features and about which no discussion is required, and that is, tuberculous meningitis. The presence of tuberculous infection elsewhere, the age of the patient, and the premonitory symptoms and lumbar puncture are usually quite sufficient to establish a diagnosis, which is often one of tuberculosis of the meninges rather than inflammation.

SEROUS MENINGITIS. There is a third type of cases, distinctly different, about which our knowledge is as yet much less complete, and they include that class known under the name of pseudo-meningitis or serous meningitis. The term meningismus has been applied to some of these cases and a simple mechanical cerebral edema is perhaps all that underlies some of them. The descriptions of this condition as given by Eichhorst, Quincke, and later by Boenninghaus and others, have caused a good deal of confusion. Quincke's original descriptions were based mainly upon 14 cases, and these were supplemented by reports of 12 more.' Boenninghaus, in 1897, collected reports of 28 cases, in each of which the patient had either been operated upon or had died, so that the actual pathological condition was known. Other cases have been reported by Hanseman, Walton, Prince, Herter, Van Gieson, Niesser, Levy and Orchansky, a total of 39. Boenninghaus makes a division of his cases into the acute and the chronic, and the acute he again divides into the benign and the malignant types. Quincke also describes acute and chronic cases.

I have made a study of acute serous meningitis following alcoholism, inanition, and the prolonged use of narcotics, and terminal stage of indulgence. In all I collected 24 cases in which autopsies were made, and in 12 microscopical examination was made of the cortex and blood-vessels. My paper was published after that of Boenninghaus, though the material was collected long before. In fact, the "wet brains" of alcoholics

give clinical pictures long known to hospital physicians and practically much like those in some of Quincke's acute cases. Still, it is the merit of Quincke to have shown that such conditions arise from various other causes than narcotic poisons and exhausted states.

According to my observation, one must probably recognize three conditions, all due to a more or less rapid serous effusion into the ventricles and subarachnoid space, and all causing symptoms like those of true meningitis. First, acute cerebral edema, usually following severe blows on the head, causing symptoms resembling meningitis, lasting two or three days, and generally disappearing with rapid restoration to health (traumatic serous meningitis). Second, the acute serous meningitis of alcoholism and allied states, called "wet brain," simulating closely meningitis, and lasting about ten days. Third, the serous meningitis described by Quincke, sometimes very acute, but sometimes subacute or recurrent and lasting three, four or more weeks. This form is still a somewhat vague one and its exact causes and pathology have yet to be worked out. It probably is oftenest due to some infection.

1. *Traumatic Serous Meningitis.* A very typical form of acute serous inflammatory effusion is seen as the result of severe injuries to the head. A person is thrown from a height or is injured in an accident and receives a severe blow upon the head, rendering him unconscious. He lies in a stupor for a day, then the neck begins to get stiff, the pupils contract, the temperature rises, the skin becomes hyperesthetic, and some rigidity of the limbs is seen, and there is constipation. He becomes a little more conscious and is slightly delirious; evidences of pain in the head are present, and the rigidity of the neck may be very excessive. There may be restlessness and twitching of the limbs. Perhaps vomiting occurs. All this looks very threatening, but within three or four days there is a change in all the symptoms; the temperature falls, the rigidity passes away, the mind gradually clears up and convalescence is established in a few days unless a pyogenic infection occurs, when a purulent meningitis develops. Both clinical observation and pathological examinations show that in these cases

there is an acute serous transudation which produces the clinical picture of meningitis.

2. *Alcoholic (and toxic) serous meningitis*, which is a sequel of profound alcoholic intoxication, and is seen occasionally in the exhaustion stage after the prolonged use of narcotic drugs, or following starvation, presents symptoms like the following: The patient may suffer at first from delirium tremens. This delirium passes away, leaving him irritable and restless, dull and irrational; or without any delirium he passes into this state as the result of a long debauch. In a few days a delirium of a mild character develops, with a slight fever of 101° or 102° F. He has but little headache or vomiting and convulsions do not occur. There is some rigidity of the neck, stiffness of the back and of the extremities, and some twitching of the muscles. The symptom of Koenig is sometimes, but not always present. The skin is hyperesthetic and the abdomen retracted. The tongue is coated and the bowels constipated. The pupils are contracted and conjunctivitis and even keratitis may develop. There is no optic neuritis. After five or six days of this excitement the patient becomes dull and passes into a semicomatose state. The neck becomes still more rigid, as do the extremities, but it is a kind of voluntary rigidity, the patient resisting movement as though every motion hurt. The skin continues very sensitive and there is a disturbance in the vasomotor system, as shown in the *tache cérébrale*. The skin loses its elasticity when stretched and lies in flaccid folds ("putty skin"). Stools and urine may be passed involuntarily. The temperature ranges from 99° to $101-2^{\circ}$ F. In the latter part of this stage there may be a sudden rise to 103° F. or more and the patient dies or begins to convalesce at about the end of the second week. In the last stage sterile serous fluid can be drawn in abundance by lumbar puncture.

The whole picture resembles closely one of ordinary purulent meningitis except that there is less headache, the onset is slower, the delirium is less acute, there is less fever, no optic neuritis, and less general constitutional disturbance. The fluid drawn by lumbar puncture is found to be clear and free from albumin or any microbes, and there is, of course, no pus.¹

¹ The symptoms of this condition are given in detail in an article entitled "Acute Serous Meningitis," *Medical Record*, 1898.

3. *The Serous Meningitis of Quincke and Boenninghaus.*² This disease is an acquired acute hydrocephalus. It occurs oftenest in children. Sometimes it runs a short course, ending in recovery (benign type), and sometimes it is rapidly fatal (malign type), and again it may become chronic (chronic acquired hydrocephalus), or the patient may get well and the disease recur (recurrent serous meningitis).

The acute form occurs in children under the age of five years in nearly half the cases, and under 30 years in three-quarters of the cases (Boenninghaus). It affects the sexes about alike. The cause is some form of infective fever in about half the cases; trauma is rarely a cause. Otitis media and sepsis are occasional factors. Bacteriological tests of the cerebrospinal fluid so far have been negative.

The disease begins with the usual symptoms of cerebral irritation: headache, delirium, followed by stupor and coma. General convulsions are frequent; rigidity of the neck and limbs, and twitchings are also present. Fever is sometimes present (one-third of the cases), but is not characteristic, neither is the pulse. Headache, however, convulsions, eye palsies and fever are not always present and it is not often possible to distinguish the disease except by its ultimate favorable course and by lumbar puncture. Even the latter procedure is not certain, because there may be some pus in the brain and none in the cord. In general, therefore, one can only say that if a young child develops symptoms of a meningitis, in the course of the exanthemata or rheumatism, or after an injury, or with an otitis, if these symptoms run a favorable course and if on puncturing the cord only a serous fluid which does not contain albumin and is sterile to culture tests, the case is probably one of acute serous meningitis. If the attack recurs, or if the child continues ill and gradually develops hydrocephalus, it may be considered a chronic serous meningitis.

The chronic form sometimes takes on the symptoms of cerebral tumor. Optic neuritis and atrophy, headache, vomiting, vertigo, convulsions, cranial nerve palsies—all may be

² Eichhorst, *Zeitsch. f. klin. Med.*, 1891, Bd. 19; Quincke, *Verhandl. der Kongl. f. inter. Med.*, 1891; *Sammlung. klin. Vorträge.*, No. 67, 1892, and *Berlin. klin. Wochensh.*, 1895, No. 41; Boenninghaus, "Ueber Meningitis serosa," Wiesbaden, 1897.

present. There may even be weakness and pains in the extremities. The chief distinguishing points are the remissions and intermissions, the increase in the size of the skull, showing hydrocephalus, and the absence of distinct localizing symptoms.

Diagnosis by Lumbar Puncture. It may be of interest here to describe the method of performing lumbar puncture. The operation is a simple one and is entirely safe if the operator is careful to perform it aseptically and not draw off more than two ounces of fluid at a time. The back of the patient and the operator's hands should be made sterile. The needle should be boiled for ten minutes. The patient should lie on the right side with knees drawn up and the uppermost shoulder so depressed as to present the spinal column to the operator. This position permits the operator to thrust the needle directly forward rather than from side to side. An antitoxin-needle, 4 cm. in length, with a diameter of 1 mm., is well adapted for infants and young children. A longer needle is necessary for adults and children more than ten years of age. The puncture is generally made between the second and third lumbar vertebræ. The thumb of the left hand is pressed between the spinous processes and the point of the needle is entered about 1 cm. to the right of the median line and on a level with the thumb nail, and directed slightly upward and inward toward the median line. At a depth of 3 or 4 cm. in children and 7 or 8 cm. in adults the needle enters the subarachnoid space and the fluid flows usually by drops but often, in alcoholic meningitis, in a spurt. It is allowed to drop into an absolutely clean test-tube, which previously has been sterilized by dry heat to 150° C. and stopped with cotton without running down the sides. From 5 to 15 cubic centimeters of fluid is a sufficient quantity for examination.

NOTES ON THREE ANOMALOUS CASES IN ONE FAMILY.*

By HORATIO C. WOOD, M.D., LL.D.

Although the cases which are recorded in this brief memoir are still alive, and therefore it is not possible to demonstrate the exact nature of the lesions, the clinical picture is in many features so peculiar that the histories have seemed worthy of record. In making such record I shall first give the details of the cases and then make my remarks upon the same.

The father of the F. family was a farmer, healthy; died at 77 of apoplexy. Mother died at 67 of cancer of the pancreas, after an illness of eighteen years with obscure abdominal symptoms, various diagnoses having been made. No nervous symptoms at all. No knowledge as to grandparents. Uncles and aunts all healthy. No nervous cases among the first-cousins.

Case number 1. This case I have never seen, the man living in California, and therefore can simply give an account as received by letter from him. The "it" of which he speaks is a pain, chiefly located on the outer side of the left leg, below the knee. Mr. J. F. says: "It came on very gradually when walking, and increased to such an extent that I was not able to walk continuously for ten minutes without stopping to rest, when the pain would almost cease, but on starting to walk again the pain would increase, and I was obliged to stop again to get relief. In this way I managed to get around to attend to my work. When sitting or lying down I suffered very little, and then only after walking some distance. The pain was a dull throbbing pain, unlike the pain of neuralgia or rheumatism I sometimes feel in other parts of the body; nor did the pain extend beyond the ankle, but when severest I felt it from the knee up to the hip. There was no swelling of any kind, nor stiffness of muscles or joints. I could press hard on the spot where it commenced, or strike it with my fist without bringing it on. Nor did rubbing have any effect when the pain was greatest. When sitting with my foot propped up as high as my head the pain decreased most rapidly. Change of weather did not affect my condition to any extent except in very hot weather, when I could walk less. In appearance there was no

*Read before the Philadelphia Neurological Society November 28, 1898. For discussion on this paper see this journal, March, 1899, p. 177.

difference between the two legs except the flesh of the leg around the affected part was softer or flabbier than the flesh around the calf of the other leg.

"After trying numerous remedies both external and internal, together with massage and electrical appliances, without getting relief, I went East, thinking a change of climate might help me, but experienced no change. [The writer of the letter lives in San Francisco, Cala.] I returned to California after four months' visit in the East, and stopping at a place where an old California stage-driver made his home, who seeing me limp along reached down in his pocket and pulled out a small bear's bone that he carried and told me to carry it in my pocket for a few weeks; which I did without giving it any thought, and to my great surprise and delight I found myself entirely free from pain in a few weeks, and could walk for several hours without any inconvenience.

"I was entirely free from pain till some nine years later, when I had the same trouble, which lasted for a year; after trying a number of doctors who made that subject a specialty, without any relief, about four months after I stopped taking medicines, and began to improve slowly; and in a few weeks was all right again, until about two years ago, when I experienced the third attack and had a new diagnosis given me, which briefly stated is that the pain is caused by the inner enlarged or varicose vein pressing on the muscles of the sciatic nerve, and that a simple operation would remove the cause, but I did not consent to the operation, and after I stopped taking medicines altogether I gradually commenced to improve and am now all right again.

"The pains lasted about a year in each case, at intervals of some eight years. I should not forget to state that I traced up the old Californian with the bone, and he kindly expressed it to me in both the latter cases, but in neither case did the bone have the desired effect. It had lost its charm."

The second case is W. I. F., aged 46. Carpenter. Single. Enjoyed good health from childhood until 1887, when he had a severe attack of croupous pneumonia. He recovered more or less imperfectly, having cough and pulmonary pain for a long time.

A few years after his attack of pneumonia a heavy wagon passed over his left leg, injuring the periosteum of the tibia, which was a long time in healing, but this injury also got well.

In September, 1895, while working in the field, he perspired freely and then sat on the ground for a short time. In the evening he returned home with a chill and fever, and symptoms supposed to be those of acute inflammatory rheumatism

developed. The pain was first noticed in the left hip and leg, and was very severe; it would appear now in the hip, then in the knee, then in the ankle. This pain would only come on towards evening. The usual antirheumatic remedies were given heavily, also free blistering with no perceptible effect. When the pain was severe in one place a little rubbing would relieve it, but it would appear instantly in another joint; and often it would shoot up the spine and locate in the back of the



Fig. I. The left knee, one inch more in circumference than the right knee, and having the appearance of an arthropathic joint.

head or over the forehead. These symptoms continued for three weeks and frequently required hypodermic injections of morphine. After the acute symptoms ceased the leg was in a helpless condition. Mr. F. could not flex the toes or leg, and there was so much weakness in the hip that he could not stand up. He managed to creep around on his hands and knees for a period of eight months. After that he could raise himself and walk by supporting himself with the back of a chair and slinging his leg forward while walking. The weakness of the back was very marked, and improved only slightly during the following year.

In November, 1896, he entered the University of Pennsyl-

vania Hospital, and was first seen by myself. His weight on admission was 207 pounds. There was then marked atrophy of the muscles of the thigh but not of the calf of the leg; he was unable to cross the unsound leg, and the patella reflexes were lost. The thigh muscles responded to strong faradic current. The tibialis anticus and peronei did not respond to faradism but did respond to a very strong galvanic current. The posterior leg muscles responded to strong faradism.

At present the left leg is very much in the same condition as it was in November, 1896. There is no distinct loss of sensibility or of the thermic sense; the thigh muscles are very much wasted as are also the muscles below the knee, especially the anterior group. The knee-jerk is absent in the left leg even on reinforcement; in the right leg it is about normal. The left thigh muscles respond feebly to strong faradic currents but give the normal reaction to the galvanic current. The tibialis anticus does not respond to either faradism or galvanism. The gastrocnemius and the peronei muscles do not respond to the faradic current; with the galvanic they give An. Cl.C. > K. Cl.C. At no time has there been any involvement of the bladder. The left knee is swollen, with rounded outline, one inch more in circumference than the right knee, painless, not tender, and having the appearance of an Arthropathic joint.

The temperature of Mr. F. is almost always subnormal, with marked irregularity, varying in the two weeks under observation from 96.5 to 98.5. Dr. Howard Mellor reports as follows concerning Mr. F.'s eyes:

"Dec. 12, 1898, vision in each eye 6-9 (?) without a correcting lens, accommodation .50 m. p. p 15 c. m. with +4. External appearances normal. Ophthalmoscope shows in the right eye: Media clear, round disc, broadened, nerve and retina over capillary, disc dirty red in color, small central physiological excavation, in which there can be seen a marked venous pulse in upper vein. Refraction, vertical vessels, +1; horizontal vessels, emmetropic.

"In the left eye: media are clear, vertically ovoid disc, upper and lower edges of disc veiled by hazy and swollen nerve fibers, these being dirty red in color, temporal third of disc grayish, nasal third dirty red in superficial layers, and grayish in deeper layers broadened scleral ring down and out. Refraction, vertical vessels, + $\frac{1}{2}$; horizontal vessels, emmetropic. The fields were taken in each eye by my assistant, Dr. Charles C. Rankin, for form and color, and found much contracted, more so in left eye than right eye, as shown in accompanying diagrams."

The third case is that of a sister, Miss L. E. F. Aged 35. Single. She was in good health until the latter part of 1897, when she began to notice in the right thumb tingling and numbness, which in a short time extended to the neighboring two fingers, and some weeks later was followed by severe pain in the right shoulder, over the scapula. At first this pain came on only at night time, waking her out of sleep, and was relieved by local hot applications. After several months the pain developed during the day, but seems always to have been paroxysmal. Some months later numbness appeared in the right leg, and later she suffered from some pain in the hip and thigh, though never in the foot. Recently there has been at times

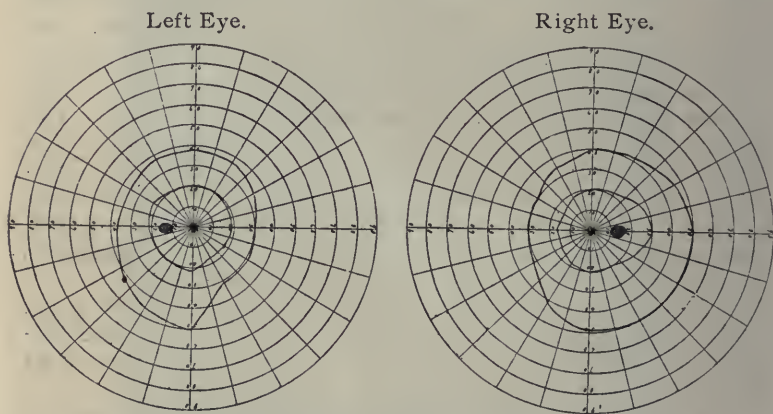


Fig. II. Fields for form and color in Case II, showing much contraction, especially in the left eye. The inner circle represents the field for red; the outer circle represents the field for white or form.

sensations of numbness in the left hand. Also, apparently in the early part of the present year, she began to suffer from motor disability in the right foot.

At the examination made October 25th, 1898, the general appearance was that of poor health but no distinct evidences of hysteria were found. Feet and hands always cold. Menstruation somewhat irregular. Urine normal. Miss F. suffers occasionally in the daytime with pain in the upper right side of the body, which, however, usually occurs in violent paroxysms at night, waking her out of sleep. The focus of pain seems to be in the right upper shoulder, above the clavicle, the pain radiating from this point into the neck and right arm, or sometimes into the left shoulder. There is also at times severe pain in the right hip. Neither active nor passive motions of the

parts affected, nor pressure over the focus of the pain, elicits more than the normal sensations. The whole right arm is very weak, and the fingers and thumb are habitually carried in flexion, suggesting but not fully conforming with the "claw-hand." The extensors are weaker than the flexors, though the latter have very little power. The thenar and interossei muscles are markedly atrophied. No tenderness can be discovered by direct pressure or by jarring or passive movement—of the vertebral column—of the joints—or of the bones, including the sternum, or of any kind of the nerve-trunks. The right



Fig. III. The hand in Case III, suggesting but not fully conforming with the "claw-hand."

wrist reflexes are abnormally active, distinctly more so than those of the left arm. In walking Miss F. has a distinct limp of the right leg, with a suggestion of spastic gait. There is marked weakness of the whole right leg, so that when lying down she cannot lift the leg in an extended position off the bed. The muscles of the lower leg, and especially those of the anterior tibial group, are, however, most affected, and there is some bending downwards of the toes. Both knee-jerks are exaggerated, but the right knee-jerk distinctly more so than the left; at times a right-sided knee-clonus of short duration is easily produced. There is marked ankle-clonus of the right foot,

none of the left. In standing there is pronounced lordosis but no lateral curvature. There is evident weakness of the muscles of the back, as shown not only by the lordosis but also by the fact that when Miss F. bends forward she can raise the body up only slowly and with the greatest effort. She is able to get up off the floor by herself without climbing over her thighs, though the movements are very slow, awkward and feeble. There is no detectable loss of co-ordination either with eyes open or shut.

Examinations made by myself on October 25th, and several times since, and also by Dr. Jenner of the University Hospital, have given varying results as to the condition of sensibility. There seems to be always a certain amount of obtunding of sensibility over the right arm and leg, but never complete loss of sensation; the points of the esthesiometer being readily recognized but not separated in a manner at all approaching the normal perception. There have been also at times partially anesthetic areas. Thermo-sensibility in several testings has been found distinctly below par but not abolished; at no time did it seem to be more obtunded than was ordinary sensibility. Electro-sensibility was about normal. The muscles of the arm, shoulder and thigh responded to the faradic current as well as the normal muscles; the response of the muscles of the leg and foot was distinctly sluggish and more so in the hand, but no distinct reaction of degeneration could be made out in the atrophied hand muscles.

The temperature of Miss F., is very irregular, most of the time, however, below the normal line; varying under observation between 98.5 and 96, and on a few occasions rising to 99.

Dr. Howard Mellor's report upon Miss F.'s eyes is as follows:

"Vision in each eye was 6-9 without a correcting lens; accommodation .37 m. p. p. 14 c. m. in right eye and 15 c. m. in left eye. External appearances normal. Ophthalmoscope showed in right eye: vertically ovoid disc, disc and retina over capillary, choroid ring (broken) all around disc, small scleral ring down and out, superficial layers of disc dirty red in color, deeper layers grayish, vessels slightly darker than normal. Refraction: vertical vessels +2; horizontal vessels + $\frac{1}{2}$.

"In the left eye, there is a vertically ovoid disc, upper nasal edge veiled, broken choroid ring all around disc, scleral ring to outside, nerve and retina over capillary, superficial layer of disc dirty red in color, deeper layers grayish, vessels toward macula region slightly tortuous. Refraction, same as in right eye. In each eye the pupils are round and react promptly to light and accommodation.

"The fields of each eye were carefully taken by my assistant, Dr. Charles C. Rankin, for both form and color and found concentrically contracted with no scotoma, as shown in accompanying diagrams."

For the purposes of diagnosis the cases just reported may be studied first, individually; second, collectively. The nature of the first case, in the absence of more detailed information, is so obscure that it is scarcely worth while to occupy space with discussion.

The lesions in the second case appear to me to be spinal and not nerve-trunkal. The earlier symptoms were believed

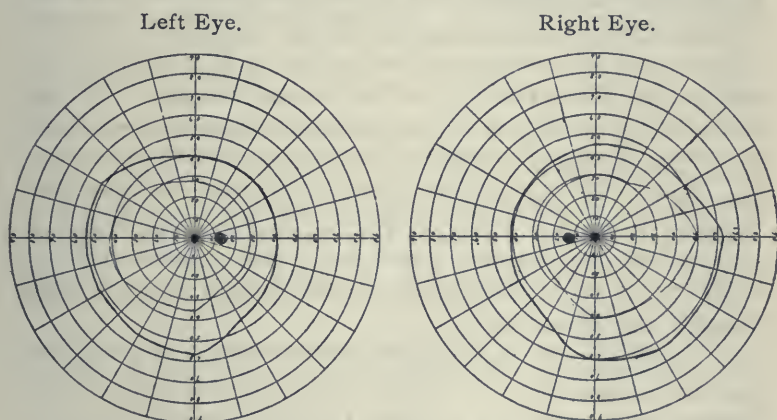


Fig. IV. Fields for form and color in Case III., showing concentric contraction without scotoma. The inner circle represents the field for red; the outer circle represents the field for white or form.

by the doctors in attendance to be of rheumatic origin, but it is noteworthy that they were not relieved at all by rheumatic treatment; and that there could have been no real tenderness of the joints, since relief was obtained by free rubbing of the affected part. I have very little doubt but that the pain was due to involvement of fibers of nerve-roots, and that the persistent lesion of a localized destruction of the nerve cells in the anterior cornua of the spinal cord.

In the third case, the first diagnosis is of course between functional, hysterical and organic nature. The marked atrophy of the muscles, the changes in the eye-grounds, the

absence of the hysterical temperament and of a history of past or present distinctly hysterical manifestations, have led me to throw out entirely the possibility of the palsy being of functional origin; and when the case was shown to the Philadelphia Neurological Society I think the verdict of all present was that it was a positively organic case. The absence of widespread pains and of tenderness of the nerve-trunks indicate that the lesion was not in the nerve-trunks; an indication which is so strongly corroborated by the heightened knee-jerks and the knee and ankle-clonus in the affected leg, and the exaggerated wrist reflexes of the affected arm, that the opinion that the lesion is in the spinal cord can hardly be avoided.

The wasting of the muscles indicates implication of motor spinal cells; the heightened reflexes point to a lesion of the conducting fibers, especially of the lateral columns. The disturbances of sensibility and especially the paroxysms of pain show that the lesion extends, however, beyond the territories just spoken of, and probably affects the posterior root-zone region. It would appear, therefore, that there is present a widespread degeneration, making the case resemble one of amyotrophic lateral sclerosis with an extension of the lesion into the neighboring parts.

In studying the cases collectively it is difficult to reach a positive conclusion. The existence of three cases in one family of four children certainly suggests some commonality of origin, but the first review of the symptoms indicates serious differences in the cases. Nevertheless, the points of similarity are very great, especially in the last two cases. The points of concordance are widespread spinal degeneration, certainly in part of similar nature, there being in each case apparent destruction of motor cells; parallelism or rather exact similarity of eye-symptoms and of optic nerve-degeneration; and habitual sub-normal temperature.

The cases are, I believe, not instances of neuritic atrophy, differing from that disorder in the history of their development, in the position and symptoms of organic changes, and in the case of Miss F., especially, in the condition of the reflexes. It seems almost impossible that there should be an exaggeration of the reflexes in neuritic atrophy. All our knowledge and

experience go to show that inflammatory changes in a nerve-trunk do not increase the tone of the tributary muscles or the activity of the reflexes. Apparently the only influence which such inflammatory nerve changes have upon the tributary muscles is one of depression, due to the more or less partial interruption of the pathway between the spinal cord and the muscles.

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246. THE ACCURACY OF VOLUNTARY MOVEMENT. R. S. Woodworth, Ph.D. (Monograph Supplement No. 13, Psychological Review, July, 1899).

This carefully-conducted and well-described research was carried out in the Psychological Laboratory of Columbia University during the college year 1898-99. It represents a very large amount of work in experimentation, as well as in compilation, and it is conducted throughout in a very commendable spirit of scientific exactness. It is especially worthy of note in that its results were prepared strictly by the statistical method, without which no research of this general nature has much value.

The voluntary movements so carefully studied were those of the arms, over 125,000 such movements having been recorded and measured in the research. The apparatus employed need not be here described, being at once simple and indisputably effective for its purpose.

The results of this research may well be taken from the table of contents, which is at the same time in part a summary. Only a portion of the points of interest to the physiologist and psychologist can here be even indicated. When the eyes are used, the accuracy diminishes as the speed increases; when not used, the accuracy varies little or none with the speed. When the speed of the motion alone increases, the accuracy of the movement is markedly in inverse ratio to the speed. When the interval between successive movements varies, the speed being constant, the accuracy diminishes as the interval is prolonged. The accuracy of initial adjustment is favored by a short interval between movements, while the accuracy of "current control" (that is, readjustments following the initial adjustment) is favored by a low speed.

As regards the validity of Weber's "law" for this phase of activity, he finds the increase in the error of movement too slow for this formula, and too fast for that law proposed as a substitute by Fullerton and Cattell. When the eyes are employed as the guiding sense-organs, the extent of the movement is judged solely thereby; when not so employed, sensations of the extent of movement are the most important factors in the control. Fatigue increases the variability of any movement; all simple central adjustments are, however, only slightly subject to fatigue. In conclusion (Part IX), Dr. Woodworth suggests, as a result of his experience in this research, an improved mode of writing based on forearm movement in place of the finger movements now in use.

DEARBORN.

ACUTE DELIRIOUS MANIA.¹

By FREDERICK J. MANN, Ph.B., M.D.,

POUGHKEEPSIE, N. Y.

LATE ASSISTANT PHYSICIAN, HUDSON RIVER STATE HOSPITAL FOR THE
INSANE; MEMBER OF THE AMERICAN MEDICO-PSYCHO-
LOGICAL ASSOCIATION.

Synonyms: Acute delirium, Acute delirious insanity, Hyperacute mania, Typho-mania, Delirium acutum, Délire aigu, Delirium grave, Phrenitis, Bell's disease, Delirious mania, Mania gravis.

Dr. Benjamin Rush, of Philadelphia, in his work "The Mind," dated 1825, speaks of mania as sometimes combined with phrenitis. He describes symptoms which we would class as fever, and says that here excitement is abstracted from the muscles and the patient usually confined to bed. He gives the name phrenia mania to it, and states it occurs most often after parturition. Luther Bell called the disease typho-mania. Regis, in his work dated 1894, says it lacks a definite position in the list of mental diseases. He considers it as the highest degree of mania, of which it forms, by its peculiar characters, a special variety. He claims it to be symptomatic, that is, connected with various morbid states as the puerperal, general paretic, alcoholic, etc. Again, it is claimed as a mode of termination of acute mania, and it is so that Clouston classes it. He places it as the third stage of common acute mania and one, he says, that does not occur in many cases if properly treated. Most authorities in psychological medicine, however, give it a distinct place by itself in the list of mental diseases.

Kellogg in his recent work states that its prompt onset, violent and brief course and fatal termination in most instances, justify the opinion that it is a distinct type of mental disease of toxic or infectious origin. This, I think, is the view most generally accepted to-day, and one that coincides with the teachings of modern bacteriological and biochemical theories.

¹ Read before the Dutchess County Medical Society, June 14, 1899.

Definition: An acute form of mental trouble, probably of infectious origin, usually of very sudden onset and course, attended with increased bodily temperature, and marked by delirium with sensory hallucinations, resembling the mental state seen in the severest cases of infectious febrile diseases; marked by incoherency, restlessness, refusal of food, general resistance to environments, loss of memory, great prostration and rapid bodily wasting; terminating in a large proportion of cases in coma and death within a fortnight of the onset of the maniacal excitement.

This trouble seems to depend on severe pathological changes, and while the frenzy, incoherency and its other symptoms may only seem to be a higher order of simple acute mania, there are bodily signs which indicate severe cerebral changes, and these are probably infectious in character.

Schüle says that the symptoms of spurious maniacal furor mark the first period of the disease, and that its second stage is that of severe cerebral exhaustion. In short, we have a condition of the most overwhelming disorder of the mind.

Predisposing Causes: The prime of life, between 25 and 40, is generally the period in which this trouble occurs. It is more frequent in women than in men, probably on account of the puerperal and climacteric states, which often bear a causal relationship to it.

Some neurotic inheritance is found in most cases, perhaps as direct hereditary insanity or as the unstable nervous organization shown by a hysterical or nervous temperament. Most cases also show some physical or nervous exhaustion from overwork or overstrain. The stresses of modern life, insufficient food, worry, intemperate habits, etc., all play a part in causation.

In considering the more active or *exciting* causes, we find that added to these predisposing conditions there is some severe moral or physical upsetting of the system. It sometimes follows fever, such as typhoid, especially if the case has been severe and with delirium. It may also occur consecutive to inflammatory diseases, as pneumonia and acute rheumatism, or after cystitis, diarrhea and diabetes. A severe alcoholic debauch may be the immediate occasion of the attack. As mentioned

before it may be induced by the puerperal state, especially where there are septic or complicated labors. This cause is not found as often as before the days of aseptic obstetrics, and apparently due to this fewer cases of acute delirium are now seen. Heat, sunstroke, injuries, thickening of the pia, surgical operations, or the effect of the anesthetic, are all to be considered. In puerperal cases, in those cases following suppurative inflammations of other parts of the body, or in acute infectious diseases, we can easily trace a source of infection.

In many other cases distorted metabolism, constipation and autointoxication are a large causative element. Mental or psychical shocks are common in leading to a mental breakdown, and in bringing on this disease—such for example as any painful emotion, fright, quarrels, money losses, deaths and griefs.

Course: Nearly always there is an initial period of mental depression of a few days' duration, and general painful feelings of mind and body are felt; there are a restless insomnia and an incipient melancholia. This initial period can be absent or so slight or short as to escape observation, and the patient become suddenly delirious; perhaps awake from sleep in that condition. These cases with sudden outbursts usually have their origin in some severe mental cause, as shock or grief, especially in those predisposed.

Now we have what may be called the active stage of the disease. This comes on suddenly, perhaps within an hour or during sleep. There is great agitation, an incessant psychomotor activity and excitement, and great expenditure of physical and cerebral forces. Accompanying this the temperature is raised, perhaps to 105° , there is a rapid pulse, growing weak, a coated dry tongue, eyes are wild with a bewildered terrified look, there is marked incoherency, both mental and motor, and exaggerated reflexes. At this stage we may have remissions of the excitement lasting a few hours, and possibly leading to some sleep.

Then perhaps improvement occurs by slow defervescence of the symptoms lasting over several weeks, and going on to a long convalescence which may lead to recovery or to a more or less demented condition of mind.

The usual course, however, is for the fever and concurrent symptoms to increase in severity, and death to occur between the fifth and tenth days. The excitement gives place to a condition of stupor or coma with typhoidal symptoms and the most extreme physical and mental depression. The patient may die suddenly from cardiac failure, or slowly from physical and nervous exhaustion. The majority of cases die in the delirious period from exhaustion after an illness of a few days; in others the excitement continues unabated for four or five weeks, stuporous symptoms increase, and the patient dies in coma.

Symptoms: There is great restlessness and muscular incoherency, purposeless or rythmical movements of arms, limbs and face, grimaces and grinding of teeth, swinging and swaying motions, and often general violent excitement. The patient appears terrified, and the eyes are wild. There is great mental excitement with rapid incoherent speech, which soon resembles a confused jargon. This speech can hardly be called delusional, so changeable is it, but nevertheless it shows a prevailing type as of fear, religious dread or humor. The patient may appear hilarious or panic-stricken. There are acute hallucinations and illusions of all the senses, of absurd or even rapidly changing violent types. In short, there is the typical picture from which the disease is properly named acute delirium. It would seem as though there is an automatic condition of mind, as though the action of the higher controlling centers is suspended; this resulting in great overaction of the lower centers. These lower automatic centers run riot, causing constant repetition of incoherent motions and speech, not of a dangerous kind but marked in frequency, number and variety, and not of such a character as an effort of the will could keep up. In this connection it might be possible to localize early the part of the brain which is most affected, according as the symptoms point to muscular trouble or to the optical, aural or other centers.

A striking tendency is that of tearing and destroying clothing, and hence go naked, or if in bed, by continual kicking of legs and throwing of arms to displace and remove the clothing. In this connection a remarkable insensibility to cold is displayed by these patients.

Blandford says that these patients do not refuse food as such, as they are not able to appreciate its nature, but oppose all attempts at care and nursing. Others claim the refusal is due to delusions, pain or spasm about the jaws, etc. Protracted loss of sleep is prominent, and what little takes place is unquiet and easily disturbed. However, some sleep—although very little may suffice—is needed to keep up life during this remarkable discharge storm of nervous energy, hence efforts to induce sleep must be earnestly persevered in. One case spoken of by Spitzka slept only one hour in eight days, then slept five hours, and from then on began to convalesce. Women seem able to withstand the loss of sleep for a longer time than men.

The temperature rises to 100, 101 or even 105 or 106 degrees. The pulse becomes weak, soft and frequent, it may reach 130, and the sphygmograph shows extreme cardiac enfeeblement. The reflexes are exaggerated at the start, but later absent. As the case progresses there are diarrhea and involuntary passage of feces and urine. Great muscular wasting and exhaustion and sordes about the mouth appear, the face is haggard and pinched, there are extreme subsultus and the other symptoms of the extreme typhoidal state. Abscesses and cellulitis are prone to occur, and would seem to be without detectable source of infection.

From October 1, 1888, to September 30, 1898, there were admitted to the Hudson River State Hospital a total of 5,359 patients, of which 58 were diagnosed as acute delirious mania, being a proportion of about one to one hundred. Of the 58, 24 died, 13 recovered, and the rest either improved or are in the hospital still; some being now in a chronic maniacal or demented state.

Case I. Admitted December 19, 1898. Female, aged 30, married, had four children; youngest, one and a half years old. Had an attack of insanity five years ago. No inherited tendencies. Cause of present attack given as poverty, overwork, and worry. Is very poor and patient's husband recently had an attack of typhoid fever. Attack began a week ago, and patient is now very incoherent, violent and delirious, has sordes on her teeth, sings, shouts, kicks and squirms. Has slept very little in past week, and is now very feeble physically. Re-

mained in about this condition with temperature ranging from 100° to 102° for a week after admission, and showed a most complete typhoidal state. Then, from what appeared to be a very slight prick of a finger, a cellulitis of the hand and arm developed, and lasted about ten days, and was accompanied by a temperature of 100° to 104°. At first patient required the protection sheet and sedatives, but in about six weeks she showed improvement and became less active physically.

The following is an example of her incoherency March 1, 1899, and is very typical of these cases: "A bottle of cod liver oil, by God, candy, black velvet, cream, my face is pretty near, hot, angel, near, I can't tell a little, stay there, cream, change it, here, now, where, hold on now," etc., this and much more being said at a very rapid rate, with a tendency for words and phrases to be repeated.

April, 1899. She was quieter, temperature was normal, she was emaciated and in very poor physical condition. Her lungs appeared weak from the great physical strain.

July, 1899. Much improved mentally and physically, childish. Helps about the ward. Will probably recover completely.

Case II. Female. Age 22. Single. Occupation, housework. Temperate in habits. Mother said to have had fits. Has been sick for some time with uterine trouble, and is in a nervous state of mind. Two weeks before admission to the hospital, the house in which patient lived was shattered by lightning, and she suddenly became violent and jumped out of the window, and seizing the first man she met told him he must kill her. Partially disrobed in presence of family physician, danced, sang and screamed. When admitted to the hospital had on handcuffs, had kicked off her shoes *en route*, and was barefooted. She claimed she was in heaven. Tongue and lips dry and fissured. Pulse 120 and feeble. Was very delirious. Was put to bed and in a protection sheet. Very noisy, delirious, weak, and in a critical condition. Temperature ranged from 99° to 101°. Had to be fed with tube. Ten days after admission required whiskey, glonoin and strophanthus. Continued in this state for several weeks, had severe diarrhea and insomnia, which required twenty-five grains of chloral each night. She received forced feeding and slowly began to gain strength and become quiet. Continued to improve and was discharged recovered seven months after admission.

Case III. Male. Age 45. Single. Employed in railroad signal tower. Hereditary influences denied. Twenty-five years ago had an attack of brain fever, which lasted three weeks and was supposed to have been caused by sunstroke. He has

worked steadily since, but has often complained of severe pain in head. Six days before admission to the hospital, while working in a hay field, he became suddenly ill, had severe pain in head and became violent and excited. However, he went to work that evening at six in the signal tower, but two hours later he became very violent, kicked down the door and smashed things in general. Was taken home, where he destroyed everything he could lay his hands on. Developed delusions of poisoning and refused to eat. On admission to the hospital was very incoherent, delirious and in a marked typhoidal state. These symptoms increased in spite of forced feeding, abundant sedatives to control agitation and free stimulation. He grew suddenly weaker and died in ten days after admission. He had two attacks of hematemesis during the six hours before death.

Pathology: First there is marked hyperemia and active congestion of the brain, which represents the excited stage of the symptoms. This is succeeded by cerebral edema with venous stasis and exudation of the blood elements, which cause the pressure symptoms. The membranes are congested, swollen and sometimes show adhesions. The elevation of temperature may be, in part at least, due to direct disturbance of the cerebral cortex or of the heat centers. The lungs are in a state of hypostatic congestion and perhaps consolidation. In fact, there is engorgement of all the internal organs. The heart is flabby, pale and degenerated, the blood thin, dark and watery, and the muscles wasted. Reports of the finding of micro-organisms in the blood have been made.

Prognosis: It is both important and difficult to make an early prognosis. The case must be considered as to whether it is to last only a few hours or days; if so only home treatment may be required, and this may be the best; or whether it is to have a duration of weeks or months, and hence to be beyond home care and demand that afforded in hospitals. Attention must be given to the character, constitution, both mental and physical, to the past history of the patient, the causes of the present trouble and the symptoms observed.

As a rule the more gradual the onset, the slower the course and ending. The severity of the symptoms must also be considered. The less the insomnia, the delirium and the incoherency, and the more and the longer the periods of compara-

tive quiet, the brighter is the outlook. Another thing to consider is the physical condition, and to what degree of severity is the system poisoned; for it requires vital force and energy to resist the products of disease.

The death rate is given by writers as between 50 and 75 per cent. of the cases attacked, and the average duration of life in fatal cases as from one to three weeks.

Differential Diagnosis: Its start helps distinguish it from common acute mania. Ordinary maniacal or melancholic frenzy follows the usual typical symptoms of these troubles, but here there is either a most sudden onset, or a short initial stage of a peculiar impaired unconsciousness. It is much more intense in the severity of its symptoms than ordinary mania. It has marked reduction of object-consciousness with delirium and absolute incoherency. There is the most utter indifference and oblivion to environments. Its course is much more rapid; and uncomplicated cases of ordinary acute mania, unless there is some contributing debility or weakness, usually tend to almost certain and rapid recovery. This is not so in acute delirium, for death is the rule in the majority of cases.

Acute Alcoholism: Here perhaps, the patient is simply mad drunk and frenzied, and if we let him sleep it off he is all right. It is a serious mistake to commit these patients to an institution for the insane, as is often done. The stigma of insanity should never be attached to anyone when not necessary. It should be a rule with every physician never to sign a commitment certificate when the person is under the influence of liquor.

Delirium Tremens: These cases are short in duration. There is a coarse tremor, a busy restless fumbling, and a different class of hallucinations and delusions. These latter are of a terrifying nature, and the patients often see all sorts of imaginary animals and objects.

In fevers, pneumonia, etc., the delirium is of a different nature, and there is also the history of the case and its special symptoms to help us. The same can be said of acute brain diseases, meningitis, etc., and here we have the special train of symptoms indicative of brain trouble, not to mention excessive pain, etc. Cases of meningitis are seen that are most difficult to differentiate from acute delirium.

Those in institutions which care for the insane occasionally see cases of low delirium of a melancholic nature. They are depressed but too delirious to be easily called acute melancholia, and still we hesitate to classify them as acute delirious mania. It is for this reason that I prefer the name of acute delirium, for it embraces all these cases. Blandford in discussing acute delirious mania mentions cases in which the patients are violent, sleepless and delirious; have religious and horrible delusions, and most obstinately refuse food, as though melancholic. This is not a mere passive resistance, but force must be used to overcome it in order to feed the patient. He says that the malady merits the name of acute melancholia rather than acute mania. Schüle speaks of a melancholia form of acute delirium, whose symptoms are said to be the very reverse of the maniacal type.

Treatment: The room should be well ventilated, darkened and cool (not over 60° F.), the windows out of reach of the patient, or else protected, and the bed single with a strong protection, or restraining sheet placed over the patient. In this way they can be kept in bed with the least violence, for of course, as in all severe cases of acute illness, a recumbent position must be insisted upon. Most careful nursing is required through the sleepless and violent stage, as the agitation must be restrained and strength supported, so that death will not occur from exhaustion.

Beware especially of the onset, when violence may come on suddenly and skilled attendants must be on hand to control the patient. Avoid the presence of near relatives as much as possible. Plentiful forced feeding is needed: Milk, eggs, minced beef, fruit juices, etc. Plenty of water should be given, or, in summer, lemonade. A pint of cream a day favors sleep and prevents constipation. Best withhold alcoholic stimulants till absolutely required, as in the latter stages, as in the early, they often increase the excitement. Chloral induces sleep and is one of the most useful drugs, especially if combined with sodium or potassium bromide. Trional and sulphonal, especially trional are of great value in bringing on sleep and controlling the excitement. Opium must be used with care, for often there is more excitement after administering it, and it

may increase the poisoning of the system; however, a full dose of morphine may at times be useful in obtaining quiet. Ergot is to be thought of early. Packing in a wet, cold or hot sheet may induce sleep, and cold sponging, full tub baths or ice packs, are indicated for the fever. A dose of calomel or castor oil early is good, and laxatives and intestinal antiseptics are of value to prevent and attenuate the autointoxication, as often there is history of more or less prolonged constipation.

247. ZUR ENCEPHALITIS PONTIS DES KINDESALTERS, ZUGLEICH EIN BEITRAG ZUR SYMPTOMATOLOGIE DER FACIALIS UND HYPOGLOSSUSLÄHMUNG (Contribution to Encephalitis Pontis of Childhood, and also to the Symptomatology of Facialis and Hypoglossus Paralysis). H. Oppenheim (Berliner klin. Wochenschrift, No. 19, May 8, 1899, p. 405).

In this case reported by Oppenheim, paralysis of the left facialis and hypoglossus developed acutely in early childhood after an attack of convulsions and unconsciousness lasting several days. The paralysis partly disappeared later and some atrophy of the affected muscles developed. In an examination made eighteen years afterwards, the muscles innervated by the left facial and hypoglossal nerves were found partially paralyzed and atrophied, fibrillary tremors existed, and the electrical irritability in the left facial distribution was quantitatively diminished. A cerebral lesion probably developed early in childhood. The paresis could not be regarded as the remains of an old hemiplegia, because the muscles affected were the ones usually restored before those of the extremities in hemiplegia. The atrophy, fibrillary twitching, quantitative diminution of the electrical response, and the exaggeration of the knee-jerk on the side opposite to that affected in the face, were regarded as contrary to the diagnosis of "fragmentary hemiplegia." Convulsions and unconsciousness lasting several days and preceding the paralysis made the diagnosis of neuritis doubtful. The case was believed to be one of pontile encephalitis—a disease which has not received a great amount of attention, although there can be no doubt that the pontile or bulbar form of infantile paralysis exists. This diagnosis would explain the exaggeration of the knee-jerk on the side opposite to the affected facial muscles. All the muscles innervated by the left facial nerve were not equally paretic, but those of the highest functional importance (orbicularis palpebrarum, orbicularis oris) had recovered their power. The fibers of the platysma myoides near the median line were active, while the lateral fibers of this muscle were paralyzed. The muscles of the facial nerve situated near the median line (mentalis, quadratus menti, orbicularis oris, corrugator) were easily stimulated by the electric current applied to the sound side—by a current insufficient to cause contraction of these muscles when applied to the left side, or distinct contraction of the sound muscles when applied to the right side. Oppenheim's explanation was that the facial muscles near the median line were innervated from the facial nerve of the opposite side.

SPILLER.

A CASE OF SUBDURAL HEMORRHAGE CAUSING ANOMIA,
WITHOUT ANY OTHER FORM OF APHASIA.*

By GRAEME M. HAMMOND, M.D.

The opinion that there is a circumscribed area in the temporal convolution in which auditory word-image memories are stored, or, in other words, that there is a "naming center," has been vigorously upheld, and supported not only by strong arguments, but by seemingly convincing pathological evidence. On the other hand, there are many who strenuously deny the existence of such a center, and equally powerful arguments are presented in contravention.

The case I desire to present for your consideration is that of a young man, twenty-five years old, of fair education, and a clerk by occupation. He denies syphilis, and there is no evidence of his having had that disease. He admits that he drinks alcoholic liquors, but says he seldom does so to excess. His physician said he had observed albumin and granular casts in the urine for several months previous to his injury. These conditions were confirmed after his admission to the hospital. He was apparently in good health at the time he was hurt. During a fight he was struck on the left side of the head, over the temporal bone, with a loaded whip-handle. He was knocked down, but was not rendered unconscious. He was able to arise without assistance, and walked to his home. He did not have any difficulty in conversing for several hours afterward. At the end of that period he was suddenly seized with a general convulsion, which from all accounts was severe and prolonged. When he regained consciousness it was observed he had completely lost the ability to name objects and persons. He was brought to the Post-Graduate Hospital a few days later, where I first saw him. I had him under observation for several days, during which time his symptoms were carefully studied both by myself and by my colleague, Dr. Joseph Collins, who rendered me valuable assistance. There were no disorders of taste, smell, vision, or hearing. There were no motor paralyses and no derangements of sensibility. There was neither word-blindness nor motor aphasia. He could talk and voluntarily say all words, except the names of objects and persons. He could say his own name readily, and did so whenever asked to. He readily recognized all objects he saw, and indicated by appropriate gestures that he understood what they were and what

*Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

they were used for, but he could not say the name of a single one, even when he was told what the name was. When shown a cup and asked if he knew what it was, he said, "Yes, sir; yes, sir," and raised it to his lips. When told to describe how he got hurt he said: "I was struck on the; yes sir; here, sir [pointing to his head], by [after an attempt to remember the name of the person who struck him] an acquaintance. He hit me with a" [here a pantomime illustrating the size of the implement he was struck with].

He could copy writing and printing, and understood both readily. When I wrote: "Put out your right forefinger and touch the third button on my vest," he read it readily and immediately complied. When told to write such words as "cat,"

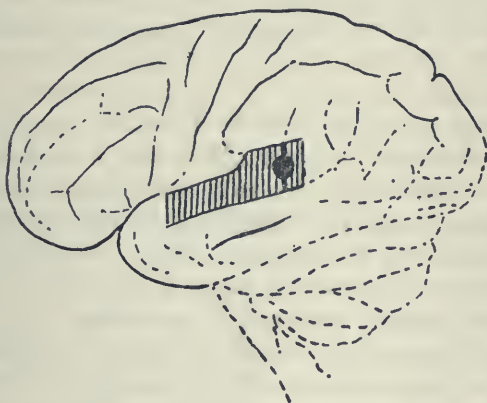


Diagram showing the location of the lesion in the superior temporal gyrus.

and horse," he wrote "crat" and "heort." He also made errors when told to say or write the alphabet. An ophthalmoscopic examination made by Dr. Collins showed slight choked disc in the right eye.

The diagnosis was made of subdural hemorrhage over the posterior portion of the superior temporal convolution.

An operation was performed by Dr. Seneca D. Powell, two weeks after the injury. I will not enter into the details of the operation now. Suffice it to say that a linear fracture was found in the left temporal bone, and that a subdural clot was found which covered the entire superior temporal gyrus. At a point at about the junction of the posterior with the middle third of this gyrus, there was a hole into which a probe could be introduced to a depth of about an inch and a half at a right angle to

the surface of the gyrus. This rupture of cerebral tissue was evidently caused by the pressure of blood, as it was certainly two and a half inches posterior to the fracture, and could hardly have been caused by the direct violence of the blow. The clot was removed. The patient made a good recovery. Four or five days later he began to make attempts to pronounce names. At first, the sounds he uttered, while wrong, bore a decided resemblance to the word he desired to say. Later he acquired greater accuracy, and now is able to recall the memory of the names of many objects and people.

It is generally admitted at the present time that the memories of word-images are stored in the posterior half of the superior temporal gyrus, probably also in the same portion of the middle gyrus, and not improbably in a small area of the inferior gyrus. As the ability to name objects and persons is a well-recognized faculty of the normal individual, it necessarily follows that there must be a cortical area in which the memories of names are stored. But does it necessarily follow that this is so invariably located in every person as to force us to recognize a certain and definite area as the cortical center for name memories? I do not believe it does. The first cells in the higher auditory center which begin to register the memory of words heard, register the memory of names. The first words the infant learns are names such as "papa," "mamma," "bow-wow," etc. These with other simple names for a considerable period of time constitute the child's entire vocabulary.

Can it be claimed that all of the cells in the higher auditory center in all infants develop simultaneously and with the same degree of perfection, and that therefore the cells in a particular and preordained part of the auditory word center are ready and prepared for the reception of name sounds and the memorizing of those words? Is it not more probable, yes even certain, that some cells are in a higher state of development than others, and that the cells which are most highly organized are the ones which will naturally register and retain the memories of names? These cells of superior development may be situated in any part of the higher auditory center. If this is the case the location of the group of cells in which name memories are stored, while being an integral part of the higher auditory

center, might differ widely in different individuals. This view is borne out to a great extent by the diversity in the situation of the lesions discovered in the cases of anomia already reported.

While it must be admitted that the conception of a word often depends upon the simultaneous combination of several perceptions, and that any interference with one or more of these perceptions may prevent the conception of a name, nevertheless there must be a distinct collection of cells which retain name memories. This collection of cells forms the beginning of the higher auditory center, but it is never anything more than a part of that center, and as its topographical position probably varies in different brains, the attempt to create a distinct naming-center seems hardly justifiable.

MOTOR APHASIA.¹

BY J. FRAENKEL, M.D., AND B. ONUF, M.D.

The paper was based upon an analysis of one hundred and three necropsies reported in medical literature. It was claimed that the evidence was strongly against the view that the pathway went directly from Broca's center to the bulbar nerve nuclei. It was probable that the insula participated in the function of speech in a similar manner as did Broca's center. Attention was also called to a test used by them to demonstrate the preservation or abolition of internal language, and which was believed to be important for the distinction between cortical and subcortical motor aphasia, especially in illiterate persons.

ANOMIA AND PARANOMIA, WITH SOME CONSIDERATIONS REGARDING A NAMING CENTER IN THE TEMPORAL LOBE.¹

BY CHARLES K. MILLS, M.D.

The details of a patient who was able to recognize objects by sight, hearing, touch, taste, and smell, but who as a rule could not name these objects, were first given. The patient, after an apoplectic seizure, developed this peculiar form of aphasia, apparently without any preceding paralysis or any

¹Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

other symptoms of focal lesion like visual blindness, hemianopsia, etc. When examined about nine years after the onset of the aphasia he had almost complete inability to name persons and objects which he was able to recognize through all his senses. He had also a marked form of paralexia or paronomia. When he attempted to read, although he understood what he was reading, he repeated an absurd formula of a few phrases. He had limited spontaneous speech; using even short sentences (without concrete nouns) correctly. He could write many single words correctly, sometimes misspelling. He held his pen or pencil correctly, and wrote such words as he did write with ease and firmness. He was not a motor agraphic. He was not word-blind or letter-blind. He could repeat a few letters (w, x, l). While the case was one of "optic aphasia," it was in reality more than this. Dr. Mills believed that the evidence was sufficient to warrant the view that destruction of a region more or less concentrated or diffused, and probably mid-temporal in location, would cause the phenomena observed in cases of anomia. While this was true, it did not follow that these speech disturbances could not be produced by lesions in other locations. Most of the regions, destruction of which would lead to inability to name persons and objects, had been summarized by Bramwell. Inability to name objects and persons might be due (a) to destruction of the "naming center," (b) destruction of the nerve fibers which passed from that naming center to the motor vocal speech center, or (c) destruction of the motor vocal speech center by which these impulses were emitted. To this enumeration he would add that anomia or paronomia with associated phenomena would also be produced by destruction of the sensory percept centers and tracts leading from these centers to the naming or concept center.

DISCUSSION.

Dr. Joseph Collins said that the subject was so very large and touched upon so many important topics that, owing to the limited time, he would have to confine his remarks to two or three points only. It was not incumbent upon him to say anything in regard to Dr. Hammond's case, although he had studied it with Dr. Hammond from the beginning. As Dr. Hammond presented the case, it was convincing. It seemed to Dr. Collins that if this case did anything at all, it absolutely contra-

dicted the existence of an uniquely localized special naming center, for here we had a sharply delimited lesion of the super-temporal convolution, occupying a location immediately adjacent to the auditory percipient and memorial center, causing exquisite anomia. It would be interesting to Dr. Collins if Dr. Mills would explain why this person was unable to name objects when the object which he recognized was brought under the percipient cognizance of the various special senses. According to the teaching of Mills and Broadbent, the naming center in the case reported by Dr. Hammond was intact. The case that Dr. Mills reported was absolutely parallel to Dr. Hammond's case. Dr. Mills stated that his patient could not recognize objects, but that should not be interpreted that he could not name objects. It did not seem to Dr. Collins at all difficult to explain the symptoms in Dr. Mills' patient by a lesion that made an interruption between the auditory center and the visual center. So far as Dr. Collins could see, the arguments in favor of an autonomous **naming center** are lacking in psychological substantiation and devoid of anatomical support, and Dr. Mills had not put on record anything more convincing in his case than he had done heretofore. Before he asks us to accept his position he should bring forth a case that will uphold it, or he should make a more legitimate plea for the existence of a naming center. When we present a case with loss of capacity to put names to objects, and show that the lesion is in the auditory center, why does not that show there is no uniquely located naming center? We do not deny that there is a naming center, if one chooses to use such a word, but we do deny the position of a special naming center in the posterior part of the third temporal convolution. There is no special area of the cortex entering into the zone of language to which the name "naming center" can be given.

Dr. Mills had referred incidentally to the case reported recently by Gordinier, and Dr. Collins wished to say, although that point was not under discussion, that that case was no more convincing than was Charcot and Dutil's case. It seemed to Dr. Collins that in Gordinier's case, which presented during life Wernicke's hemianopic pupillary reaction, there must have been something more anatomically than was recorded.

Finally one word in regard to Drs. Fraenkel and Onuf's paper. Dr. Collins was unwilling to concede that it was any adequate test of the existence or intactness of the internal language to be able to pick out the fifth card as described by Drs. Fraenkel and Onuf. One could teach a parrot to do that in five days. In fact, it is just what the Italian's fortune-telling canary does. Dr. Collins would like very much to hear Dr.

Onuf's explanation of the psychical antecedents of picking out these cards, and wherein it required the existence of internal language. The Proust-Lichtheim test had serious limitations of application, but it seemed to Dr. Collins far better than the test of Fraenkel and Onuf.

Dr. E. D. Fisher said that if we ask our patients how they recollect a thing, some will tell us that they remember it best by associating it with the written picture, and others that they remember it best as they have heard it. We find this constantly in our study of children or in teaching of languages, etc., and this may have some bearing upon the question as to whether we shall find one fixed point for the naming center in the brain. Dr. Fisher did not see why we should expect to find such a center. While the case of Dr. Gordinier was a very important one, he would not, on the same grounds, expect to find that we have a center for writing. It does not settle the question any more than the cases that have been brought to our attention during the discussion had decided that there is a definite naming center.

Dr. W. G. Spiller agreed with Dr. Onuf that we must give up the word "subcortical" in speaking of aphasia, as the case published by Dejerine and Sérieux had shown that the so-called subcortical sensory aphasia may result from a cortical lesion, and it seemed probable that pure motor aphasia might also result from a cortical lesion.

He had been discussing with Dr. H. M. Thomas, Drs. Fraenkel and Onuf's method of making out tracts and centers, by putting together the diagrams representing the areas of greatest degeneration in all the cases studied by them and determining from these the location of tracts and centers. The method was open to criticism. Many of the cases were studied macroscopically only, and important areas might have existed in tissue whose degeneration could only be detected by the microscope. If the cases studied macroscopically confirmed those studied microscopically the results would be more valuable. It must be remembered, however, that an important tract might be in an area only slightly degenerated, and yet the function of the tract be destroyed.

Dr. Spiller asked in what way, in testing pure motor aphasia, Drs. Fraenkel and Onuf's method with cards was superior to that made so well known by Lichtheim, and consisting in pressing the hand of the observer by the patient as many times as there were syllables in the test word.

Dr. Onuf replied that the method suggested by Dr. Fraenkel and himself was intended for illiterate persons to whom the method mentioned by Dr. Spiller was not applicable.

Dr. P. C. Knapp said he would hardly be willing to accept Dr. Mills' naming center, but he must object to the opinion brought forward by Dr. Hammond and Dr. Collins on the strength of the case reported by Dr. Hammond. The general experiences in injuries of the brain is that you cannot be certain of the lesion by considering it through a small trephine hole, or even a large hole. We know that in these cases there are often extensive lesions of the brain apart from the immediate site of the hemorrhage, and Dr. Hammond said that the hole in the temporal lobe was two inches or more in size, so that there may well have been contusion or laceration of a considerable portion of the brain beyond the site of the lesion.

In regard to the writing center, there was a point that Dr. Knapp thought had not been dwelt upon, and that is, that if there be a writing center, as he was inclined to believe, it is the last center to be evolved, and of course it does not exist in a great number of people, because only a small number of the human race are able to write at all. In a very large proportion of those who are able to write all the muscles of the body assist in writing—that is, the whole brain cortex, and not a small portion of it, is put into action.

Dr. Hammond said he had little to add except in reply to Dr. Knapp's criticism. In the operation in his case the bone was removed after several trephine-openings had been made and the communications sawed through with the electric saw, so that a piece of bone was removed of sufficient size to expose completely the fissure of Sylvius and the brain tissue about it. Several careful observers, as well as Dr. Collins and Dr. Hammond, saw the fissure of Sylvius and the superior temporal convolution, and located the different portions by careful experiments. A small hole was found in the brain substance that admitted a probe which passed directly in, a distance of an inch and a half. An important point which was worthy of mention was, that on the day following the operation the patient showed evidences of sugar in the urine, and the sugar was present for a few days only.

This was a well-marked case with a very definite lesion, and the man had anomia pure and simple. It seemed to Dr. Hammond that the situation of the so-called naming center was in this particular case clearly marked out, but it does not follow, by any means, that the naming center need be in the same position in every case. The naming center must be the nucleus of the higher auditory center. The child begins to learn names, and those names may be registered in the superior convolution in one case, and somewhere else in the higher auditory center in another case.

Dr. Onuf heartily coincided with Dr. Collins' view that we cannot speak of a naming center, and he believed that the function of naming an object is not a function of *one* region of the brain, but that several centers participate in it. This is confirmed by the behavior of the patients. Their principal defect lies in not being able to name *objects* either from sight or touch or other senses, and in the great difficulty of using names of objects in voluntary speech to convey their meaning. They usually describe the use of objects instead of naming them directly; otherwise they speak rather fluently, and this proves that it is not so much the naming faculty *in general* that is concerned, as that of naming *objects*. Moreover, these patients have no difficulty in repeating any names (also of objects) told them, and can usually also utter them in reading. This shows that the memories of names themselves are not destroyed, and that only their relation to other functions is disturbed. Dr. Onuf was, therefore, certain that the condition was due, not to the destruction of some center, but to the interruption of some connecting pathway between two or more centers, and especially the connection between the center of the auditory word images and the center of the visual memories of objects.

Dr. Collins had asked what association of ideas a man has in making the test mentioned by Fraenkel and himself. Probably Dr. Collins had misunderstood the meaning of the test. It was claimed that the patient is, according to the plan proposed, forced to make use of internal language, namely of counting in words. It was believed that if the test were applied with proper discrimination, the use of visual images by the patient, which of course would nullify its value, could be prevented.

Dr. Mills said that even his friends who opposed him were coming to the point of view that there was a naming center. The only difference seemed to be that they had half a dozen naming centers. Bramwell said it was a component part of the auditory center. Just so long as there were concepts, just so long must there be higher centers than the auditory percept center for their origination. This region was a higher evolution of the zone of language, and had been formed in the process of evolution.

Periscope.

PATHOLOGY.

248. BEMERKUNGEN UEBER DAS VERHALTEN GEWISSEY ORGANE GEGENUEBER SPECIFISCHER INFECTIONEN (Observations Concerning the Relation Between Certain Organs and Specific Infections). v. Babes (Berl. klin. Woch., No. 17, 1899).

This paper is a continuation of some work published in 1889. In that paper the author enunciated the fact that normal nerve substance injected into animals with hydrophobia counteracted the effects of that infection. In a later paper he showed that the gall of a rabid dog mixed with the virus of hydrophobia prevented the development of rabies in an infected dog. He concludes from this that in the incubation stage of the disease a substance which normally exists in the nervous system is eliminated from the system through the gall. In a long series of experiments on dogs and rabbits he found in the incubation stage of the disease, when no symptoms, or only slight fever, were present, well-defined changes in the nervous system. These consisted of proliferative changes in the walls of the vessels and chromatolysis of the ganglion cells of the cord and bulb. These changes were first noted around the central canal. The poison, therefore, penetrates the cord from the central canal, producing edema, leucocytosis, irritation of connective tissue, and degeneration of the nerve cells. The poison is distributed along the nerves, and affects the motor centers. The nerve cells attempt to paralyze the effects of the poison by the elimination of their chromatic substance, and so long as sufficient cells remain which do not show this change, symptoms do not develop and the disease does not advance beyond the incubation stage. The liver, therefore, reacts differently, depending on the character of the infection. It may act as a filter, and thus separate the micro-organisms, or it may secrete some material which has a bactericidal action. This substance may pass into the blood and protect it from infection, as in typhoid. Finally, the liver can assist, through the gall, in the elimination of the neutralized virus.

McCARTHY.

249. UEBER VERÄNDERUNGEN DER NERVEN BEI ACUTER STÖRUNG DER BLUTZUFUHR (Concerning Changes in the Nerves Caused by Acute Disturbance of the Circulation). M. Lapinsky (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, Nos. 5 and 6, p. 364).

A number of cases have shown that when an artery is suddenly closed, symptoms of nerve involvement rapidly develop. Lapinsky reports six cases of this character, and proposes the name of neuritis ischæmica for this form of nerve degeneration. From a study of his cases and of others in the literature he thinks:

Ischemia of the extremities lasting a few days can cause disease of the peripheral nerves.

Motor function is impaired, and soon entirely abolished.

Sensation (for touch, pain, location, heat, and cold) is impaired and soon lost.

Skin and tendon reflexes are soon weakened, and then lost.

Electrical irritability of the nerves to the faradic and galvanic currents gradually diminishes, and is soon entirely lost.

The anatomical changes of the nerves consist of swelling of the connective tissue, disintegration of the myelin, and some increase in the number of the nuclei of the sheaths of Schwann. The axis cylinders become cloudy, sometimes disintegrate and entirely disappear.

If a collateral circulation is speedily established improvement, or even recovery, occurs in the nerve functions.

The muscles are less involved than the nerves.

SPILLER.

250. *UEBER MYELITIS ACUTA (Concerning Acute Myelitis)*. H. Hochhaus (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, Nos. 5 and 6, p. 395).

Acute myelitis has been believed by Oppenheim to be a rare disease, but four cases observed by Hochhaus within two years would seem to indicate that it may not be so exceedingly uncommon. The duration of the process in these four cases ranged from twenty-six days to two months and twenty-five days. The clinical course was not abnormal. The prodromal symptoms—chiefly pain in the arms and legs—were short, and in one case were absent. Paralysis developed very quickly, *i. e.*, within a few hours or two days. Death occurred from pulmonary or nephritic complication. The cause in one case was cystitis or pyelonephritis, in another it was probably acute tonsillitis. Hyperemia, perivascular cellular infiltration, and small hemorrhages were found. These changes extended throughout the cord; in one case into the brain, and in another to the medulla oblongata and pons. Swollen axis cylinders were rare, but fatty, granular cells were numerous. The neuroglia cells were in places increased in number. The gray matter escaped to a remarkable degree, except in one case. Meningitis, of different intensity, was found in all the cases, and in two cases foci of softening were found in the posterior columns. The peripheral nerves and muscles were examined in one case and found diseased.

SPILLER.

251. *UN CASO MORTALE DI COREA DEL SYDENHAM CON RICHERCHE BACTERIOSCOPICHE ED ISTOLOGICHE (A Fatal Case of Sydenham's Chorea, with Bacteriological and Histological Examination)*. G. Daddi and R. Silvestrini (La Settimana Medica, July 1889, No. 29, pp. 337-341, and No. 30, pp. 351-353).

This case deals with an unmarried domestic, of 17 years, whose previous history had been good up to the age of 12, when her mother died of some chronic pulmonary trouble. The family history was good from a nervous and mental point of view, excepting for the fact that her brother had convulsions at the age of 4 and became hemiplegic. After the death of her mother the patient developed an eczema of the scalp, which was followed by permanent enlargement of the cervical glands. At 16 she was seized with severe pains in the right leg and the latter became swollen, especially in the neighborhood of the knee and ankle. She was not confined to her bed, and the trouble disappeared in about three weeks. After this she had another attack of eczema, which soon got well under treatment, but was followed by renewal of the pains in the right leg and by slight palpitation of the heart. Twenty days before coming under the author's observation the patient began to exhibit choreiform movements, especially of the right arm and leg, which later involved the cervical and facial muscles, and ultimately the left side. These movements finally became so severe that the patient could not lie down; alimentation was with difficulty secured, and her speech also suffered. There was marked evidence of hallucinations of sight, and she spent much of her time before an open window

in order to get relief from an annoying sense of heat. Just before her entrance to the hospital there was loss of sphincteric control. On entrance the choreic movements were found to be general, but more marked on the right side. Speech was unintelligible, and there was much complaint of headache. Examination of the internal organs was negative, except for a slight increase in the transverse diameter of the heart and impurity of the first sound at the apex; the urine was free from sugar and albumin. Temperature, 38.3° C. on entrance, gradually rising to over 40° C. In spite of active treatment with bromide, chloral, and strophanthus, the condition of the patient grew progressively worse, and on the fourth day the legs seemed to be in a condition of flaccid paralysis. On the seventh day, during the period of maximum temperature a puncture was made and a few cubic centimeters of a perfectly clear, thin fluid obtained. Death occurred during the evening of this day, after a short period of coma. Cultures from the fluid obtained by the lumbar puncture were made in various media, some of which were injected subcutaneously into white mice; two rabbits also received small doses subdurally. The necropsy was made two days after death, the body having been carefully preserved on ice. Nothing microscopically abnormal was detected in brain or cord. In both thoracic cavities were fibrous pleuritic adhesions, and hypostatic processes in the base of both lungs. The heart was of normal dimensions, but showed a typical verrucous endocarditis with slight vegetations surrounding the mitral orifice, the other orifices and valves being free. The liver showed incipient fatty degeneration; the spleen parenchyma was soft and of the color of wine dregs. The kidneys were normal in volume, and the capsule not adherent. Digestive tract normal. Examination of the central nervous system, peripheral nerves, liver, spleen, and endocardium showed no micro-organisms. No hemorrhages were noted in the meninges, but the vessels of the pia were markedly hyperemic. In the frontal region of the brain marked changes were found in the Nissl substance of the cellular elements, the part coloring with thionin being reduced to several small granules, which were either scattered throughout the cell-body or gathered together at the periphery, while certain cells had entirely lost their chromatic substance, the protoplasm appearing coarsely granular. Many of the cells themselves, notably the large and medium-sized pyramidal, now so swollen as to lose their pyramidal form and had become bottle-shaped; and in certain of them there was peripheral dislocation of the nuclei. In sections colored with picocarmine the cellular protoplasm stained but faintly throughout, giving rise in places to an appearance of vacuolation. No hemorrhages were seen, but the vessels were very turgid. By Golgi many elements were seen which showed varicose enlargements of their protoplasmic prolongations. In the psychomotor zone the same changes were found as above, but less intense. The gray matter of the temporo-sphenoidal lobe appeared (on section) as if riddled by minute holes, and these vacuoles seemed to have their walls made up of thickened interstitial tissue; they were not in the neighborhood of vessels. The cellular elements showed the same marked changes as in the frontal region, and hyperemia was also marked. In the occipital lobe the same condition existed, but to a less marked degree. The cerebellum and bulb showed no very decided variations from the normal. The cervical cord presented nothing but slight changes in the amount and arrangement of the chromatic substance of the anterior and posterior horns and a granulation of the protoplasm, and the same condition, though less intense, could be traced down through the dorsal lumbar regions. In the spinal ganglia marked variations were found in the disposition of the chromatic substance of the various cellular elements, even in the

same section. In certain cells this substance was found to be entirely normal, in others somewhat diminished, in others still it appeared as a very fine powder, and in certain ones it seemed to have entirely disappeared, the cell staining homogeneously. Pigment in abundance was found in the elements of the entire nervous system. In the cord there was intense hyperemia of all the vessels, even to the most minute; the perivascular lymph spaces were not visible.

The authors conclude their article with a review of the literature on the pathology of chorea and the establishment of an hypothesis as to the genesis of this disease.

J. W. COURTNEY.

CLINICAL NEUROLOGY.

252. BEITRAG ZUR CHRONISCHEN ANKLYLOSIRENDEN ENTZÜNDUNG DER WIRBELSÄULE UND DER HÜFTGELENKE (Contribution to the Chronic Ankylosing Inflammation of the Vertebral Column and the Hip-Joints). Valentini (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, Nos. 3 and 4, p. 239).

Valentini reports two cases of spondylose rhizomélisque, in one of which, contrary to the usual rule, some of the small joints were involved. An important distinction between this disease and chronic articular rheumatism, or arthritis deformans, is, that ankylosis occurs in every case of spondylose rhizomélisque, and the rigidity in chronic articular rheumatism is scarcely ever complete, and is due to pain and swelling of the capsule of the joint. The characteristic features of spondylose rhizomélisque, according to Valentini, are found in the ankylosis of the affected joints, in the beginning of the disease in the vertebral column, in the secondary involvement of hip and knee joint, in the almost invariable escape of the small joints, and in ankylosis of the small joints when they are attacked. The nervous system remains normal, and in this spondylose rhizomélisque differs from v. Bechterew's rigidity of the vertebral column. Valentini distinguishes four articular diseases having resemblances to one another, viz., chronic articular rheumatism, arthritis deformans, spondylose rhizomélisque, and the rigidity of the vertebral column with intercostal neuralgia (v. Bechterew).

SPILLER.

253. CHIRURGISCHE EINGRIFFE BEI HYSTERIE (Surgical Measures in Hysteria). M. Sander (Deutsche med. Wochenschrift, Sept. 7, 1899, p. 588).

Sander reports two cases of hysteria which simulated disease of the intestinal tract, and in one case operation was performed twice; in the other, four times within a year. The first patient presented more the symptoms of intestinal obstruction; the second more those of perforation peritonitis. The ileocecal region seemed to be especially involved. The chief sign in the first case was meteorismus, occurring frequently with pain. This was first seen after a hysterical attack, and the diagnosis of hysteria was at once made. Variation in the frequency of the pulse, and later vomiting, fever, and a painful resistance in the ileocecal region were observed, and the diagnosis became doubtful. The vomiting was regarded as an undoubtedly hysterical sign; the increase in temperature was either artificially produced or was the result of constipation, and the latter seemed more probable, as the temperature did not go much above 38 degrees C., and usually fell with action of the bowels. The pain on pressure in the ileocecal region was probably in a hyper-esthetic zone. The operations revealed no organic disease.

The second patient came to the hospital on account of pain in the right hip, and the diagnosis of hysterical coxalgia was made. She was desirous of arousing the interest of the physicians. Later she had frequent vomiting, distended abdomen, diarrhea, and some tenderness of the abdomen, at first especially in the ovarian region. The diagnosis of *ulcus ventriculi* was made. Circumscribed tenderness in the region of the stomach, and a tender point to the left of the vertebral column, and blood on one occasion in the vomit, seemed to strengthen this diagnosis. Fever developed, but the disproportion between the fever and the pulse rate was striking. Perforation and circumscribed peritonitis were believed to have occurred, especially as dullness in the region of the stomach with pain and loss of strength were observed at this time. The dullness disappeared after a few days. Nourishment was given by the rectum until it was observed that food taken secretly by the mouth caused no bad symptoms. Operation revealed no sufficient cause for the symptoms.

SPILLER.

254. UN CASO PARAPLEGIA DA PERTOSSE (A Case of Paraplegia from Pertussis). Ezio Luisada (*La Settimana Medica*, Jan. 1899, No. 3, p. 25).

In the above case Luisada makes a very interesting and valuable clinical contribution to the study of cord lesions arising in the course of whooping-cough. The patient was a female child of five years, of tardy development, and of not altogether good nervous heredity. She had had enteric disturbance in the first months of life and measles in the seventh, which left her with a bronchial catarrh that persisted for four or five months. During her fifth year she contracted what at first seemed a mild type of pertussis, but which subsequently became so severe as to cause conjunctival ecchymoses from the violence of the paroxysms. Soon after the appearance of these vascular disturbances it was noticed one morning that the child, on getting out of bed after a night not characterized by any special rise of temperature or unusual nervous phenomena, was very weak and unsteady on her legs, and that she complained of pains in the latter. During the day the pains increased in severity and the weakness of the legs went on to the establishment of complete paraplegia; there were also girdle pains, and tenderness on pressure over entire spine below the upper dorsal region. The child had great trouble in starting her water, and the desire to urinate persisted after the bladder was fully emptied; constipation was marked. There was no vomiting and no cranial nerve trouble. After a few days slight improvement was noted in the pain, and the paralysis had decreased somewhat. Two months later examination showed head and upper extremities free from trouble, and nothing abnormal in the respiratory tract except some sibilant râles in the right back, and a slight emphysema. The abdomen was humid, with flaccid muscular walls, and the inguinal glands were somewhat enlarged; the abdominal reflex present, but weak. There was tenderness on pressure over the spine, especially in the dorsal region. The legs were of normal volume and consistence on both sides, but there was almost absolute paralysis of all muscles, and the adductors of both thighs were evidently contracted; the feet showed slight plantar flexion. Attempts at passive movements of legs encountered resistance in all muscles. The patella and plantar reflexes were very lively and ankle clonus was slightly present, especially on the right. Tactile sensibility was retained on lower extremities and abdomen, but painful impressions were lost in these regions up to a line encircling the body on a level with the umbilicus; above this

hyperesthesia was marked. Walking was entirely impossible, and on being supported the child held the lower limbs in a condition of marked rigidity. Electrical irritability was preserved throughout, except in the rectus and obliquus abdominalis, especially the rectus, where some quantitative diminution to both currents was found, but no polar changes. The treatment consisted of KI internally, and the application of the galvanic current. For two months there was persistence of sphincteric trouble, pain and muscular rigidity, with excessive sweating day and night. Then improvement began, which ended in complete recovery four and a half months after the onset of the disease. The child could then run, jump, and perform all movements as before her sickness, but there still remained a slight varus of the right foot, and the latter was dragged somewhat in walking. From the suddenness of onset of the symptoms and their gradual increase during the first days of the disease, the author favors the diagnosis of hemorrhage, and from the rather short duration of the process he inclines to the belief that the bleeding occurred in the meninges, rather than into the cord. He does not positively exclude cord hemorrhages, however, because in a case of pertussis which came to autopsy from the pediatric clinic in Florence there were found minute hemorrhages in the spinal cord, medulla and brain, which had given rise to no discoverable symptoms *intra vitam*. Luisada was unable to find but one case similar to his in the literature.

J. W. COURTNEY.

255. BEITRAG ZUR CASUISTIK DER OPHTHALMOPLLEGISCHEN MIGRÄNE (Clinical Contribution to Ophthalmoplegic Migraine). Rudolf Paderstein (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, Nos. 5 and 6, p. 418).

Paderstein reports two cases of ophthalmoplegic migraine, one of which was rather atypical, and gives abstracts from a number of cases in the literature in support of his views. He believes that there is an idiopathic disease in which migraine is combined with attacks of paralysis of ocular muscles. The name of "periodic, or recurring oculomotor paralysis," as applied to this affection, is objectionable, because the prominent symptom of migraine is ignored, and the involvement of ocular muscles not supplied by the oculomotor nerve is excluded; the name of ophthalmoplegic migraine is, therefore, better. It is uncertain whether the ophthalmoplegic migraine is a variety of ordinary migraine or a disease *sui generis*, as the etiology of both forms is unknown.

SPILLER.

ANATOMY.

256. THE NEURON RETRACTION THEORY (Weil, R., and Frank, B.).

We are indebted to Dr. Ira Van Gieson for a synopsis of a paper by Mr. Weil and Mr. Frank on the inadequacy of the evidence of neuron retraction furnished by methods of the Golgi type, which was read before the New York Pathological Society on work done at the "Pathological Institute."

Investigation is directed as to the validity of the occurrence of varicosities and retraction of gemmules along the dendrons as evidence of retraction, as demonstrated by the Golgi methods. The investigations were based on 43 cases; five were of human material, the rest rabbits; ten of the rabbits were normal, two were poisoned by morphine, one by strychnine, four by chloroform, and the rest by the injection of hypertoxic urine, or serum.

Other investigators have uniformly employed either the rapid Golgi (or Cajal) method, or the Cox method. In this research nine of the cases were treated according to four separate procedures, namely, the slow, mixed and rapid modifications of the bichromate of silver method, and the Cox modification of the bichromate-sublimate method; two cases, by the rapid method and that of Cox, and the rest of the material according to some one of these procedures alone. A comparison of this material with especial reference to the alleged pathological changes demonstrated the following points:

First, that the same material when treated by the different methods yields different results; thus, all material treated by the slow modification shows dendrites which are uniformly non-varicose, while any material treated by the other three procedures regularly reveals varicosities on the dendrites. Second, the above results are independent of the nature of the material, whether derived from normal or poisoned animals. Third, the same material does not yield constantly identical results when treated either by the mixed, rapid, or Cox procedures, as fragments cut from the same cortex, and immersed in the same fluid, of any one of these three methods may show dendrites with every degree of the varicoseness.

JELLIFFE.

257. DER GEGENWÄRTIGE STAND DER NEURONLEHRE (The Present Position of the Neuron Theory). A. Hoche (Centralblatt f. Nerveneheilkunde u. Psychiatrie, 22, 1899, p. 276).

Hoche is one of the school that would teach the passing of the neuron theory. He here presents a series of conclusions in which he writes that the conception of the neuron as outlined by Waldeyer is no longer tenable. The fibrillar theories of Apáthy, Bethe, and others do not interfere with the data derived from embryology. The individuality of the neuron in a histological sense is no longer to be upheld for vertebrates. Human and animal experimental pathology show that there is an individuality in the neuron, trophic and functional, even if the evidence for histological individuality is wanting. The author thus tries to make incomplete histological evidence of more value than complete physiological truth.

JELLIFFE.

258. UEBER CENTRAL NEURITENENDIGUNGEN (On Central Nervous Endings). Semi Meyer. (Archiv f. mikroskopische Anatomie, 54, 1899, p. 296).

The old Gerlach reticulum about the cell-body has still a place in nerve cell histology, according to Meyer, who here contributes a critical study of the nerve terminations within the central nervous system. Very recent work by Golgi and Cajal by the silver methods bears out the testimony afforded by the methylene blue method, and seems to show that the nerve terminations about the cell body are extremely complex. The nerve termination comes in contact with the cell body, spreads about it, and completely enfolds it. He also maintains that the present-day conception of the Waldeyer neuron is tenable, and that Apáthy's and Bethe's evidence, even if mainly authentic, does not necessarily negative the generally accepted view of the neuron theory; moreover, continuity of fibrils is as yet only conjectured, not proven.

JELLIFFE.

PSYCHOLOGY.

259. "NOT THE DISEASE ONLY, BUT ALSO THE MAN," James J. Putnam, Shattuck Lecture, June, 1899, p. 33.

This lecture was delivered before the Massachusetts Medical Society at its last annual meeting, and it is now reprinted in pamphlet form. Dr. Putnam does much more in this article than to reiterate the exhortation (so frequently, but never too frequently, heard) expressed in its title, for he tells how the "man" is to be treated, especially in those very numerous cases in which the disease has a so-called "mental" or neurotic aspect. It is a timely article, for it at once explains and advocates the employment of about all that is of value medically in hypnotism, "Christian Science," faith-cures, etc., namely, therapeutic suggestion. As physician to the nervous department of one of the country's large hospitals, Professor Putnam has had ample opportunity both to elaborate theoretically and to put in practice what he means, as indicated in this lecture. It is refreshing to meet with a discussion of disease which looks into man with a seeing at once more penetrating and broader than that which the all-too-practical physician is apt to employ. "The man," says the lecturer, "is, above all else, the mind of the man, and not only the mind as an organ of conscious thought, but the mind as an organ of bodily nutrition, and the mind as a vast theater for the interplay of contending forces that do not always recognize the personal consciousness as their ruler." * * * "I should rate a thorough preliminary course in psychology and philosophy far above a knowledge of botany or zoology, and as following close on chemistry and physics as a preparation for the work of a general practitioner. The physicians who have previously been good students of psychology have, in my experience, proved themselves to be men of unusually broad sympathies and high ideals, and keen to see causes of disease to which the pure pathologist is often blind. No argument is needed to show what transforming power the mind may exert. The energy set free by the magic agencies of hope, courage, desperation, fanaticism, or by the enthusiasm for a great cause, may reveal the possession of a force undreamed of, or so husband the resources of the body as to keep the flame of life burning for a time when the oil seems exhausted." The physician is, properly speaking, by his profession an educator, obviously enough of children, but likewise properly of adults as well, and above all, of that great multitude whose lives needlessly are damaged by certain forms of disease, especially those known as habit neuroses, in which in some degree or other so very many share. It is these that suggestion will control and cure, and its field of application is indefinitely wide. It means nothing to tell a person suffering in mind or body that his "pains are imaginary"—psychology recognizes no such thing as imaginary pain. What is demanded in these cases is persistent encouragement, accompanied by an explanation of his condition that has the force likewise of encouragement, leaving in his mind no doubt of the physician's convictions. If these means fail, coaxing or training will show the hypochondriac how idle his notions are. A more concentrated preparation of this medicine (universally employed by everyone in his dealings with his fellows, as influence), a form about which much has been written of late, but which is rare as yet in therapeutics, especially in America, is suggestion under actual hypnosis. Only now is it being learned that oftentimes "waking suggestion" is all that is necessary, as indeed the faith curists and the trusting readers of the "Bible Annex" (as Mark Twain has named a certain Mrs. Eddy's book) have long since discov-

ered. In cases where waking suggestion is too weak, hypnosis, thinks Professor Putnam, has excuse.

Such, then, are the increasing degrees of suggestion often beneficially applied to disease. Whichever is employed, the treatment of these numerous cases of diseases of association and of habit must follow one of two familiar courses: The first is that of eliminating from the mind of the patient the troublesome and ever-recurrent impulse or habit; the second, that of introducing another impulse incompatible with the other and tending, therefore, to its exclusion. Of these the former is usually the more successful, and the one which really does the work even when it is ostensibly not employed. In this way recreation, rest, physical exercise, literature, exert a tremendous influence, helping along that which should be the physician's constant aim—to get the sufferer out of his psychophysical habit, out of his wretched self.

It is in this far-reaching recognition, both as a theoretical concept and as a practical principle, of the perpetual unity of the psychophysical individual, that the force and usefulness of Professor Putnam's method lies; the body and the mind of a man are not two, but one, and of this basal fact the practitioner of medicine, seeking ever to relieve and cure, cannot be too often reminded.

DEARBORN.

260. ON THE INVALIDITY OF THE ESTHESIOMETRIC METHOD AS A MEASURE OF MENTAL FATIGUE. Geo. B. German, M.D. (*Psychological Review*, Vol. VI, No. 6, September, 1899, pp. 599-605.)

This article is an empirical denial of a statement of Dr. Griesbach (1895), to the effect that there exists a close and definite correspondence between the extent of sensation areas and the fatigue incident to mental work induced at school and elsewhere, the main hypothesis being that fatigue increases the size of these areas, while rest diminishes their extent over any portion of the skin. Dr. German doubted the validity of the previous experimenter's method, that, namely, of increasing the minimal distances and decreasing maximal distances between the esthesiometer points until the extent of the sensation areas was in this way determined, one determination only being apparently made in each case.

Dr. German's subject was his sister, a Barnard student, aged twenty-three, and sound in body and mind. He used the psychological method of right and wrong cases, and Jastrow's esthesiometer was the instrument employed. The experiments (about 2,450 separate discrimination-judgments were made) were conducted during thirty days, about half being between eight and ten in the forenoon, and the remainder between nine and quarter-after-ten at night, the intervening time being employed in labor amply sufficient to produce a proper degree of normal fatigue.

The net product of the little research is interesting and suggestive of several things, and may be stated in the author's own words: "In at least one normal instance the percentage of errors in cutaneous tactile discrimination bears no constant, nor even relative, correspondence to the mental fatigue experienced by the subject."

DEARBORN.

Book Reviews.

- E. BRISSAUD: LEÇONS SUR LES MALADIES NERVEUSES. Deuxième série. Recueillies et publiées par Henry Meige. Avec 165 Figures dans le texte. Paris: Masson et Cie, Éditeurs, 1899.

This second volume of clinical lectures by Brissaud could with propriety be called a contribution to spinal localization. The two important topics most fully treated are the spinal metameres and the syndrome of Brown-Séquard. The work, to be sure, is almost entirely clinical, and in this respect is too much lacking in a sound histological basis to be accepted as conclusive; but the author's admirers, especially in France, will, nevertheless, probably accept it as a demonstrative work. It abounds in diagrams and schemes, with which French writers are especially fond of decorating their pages, and which lend themselves so conveniently to the illustration of sensory changes on the skin. We confess that for our own part we should prefer to have more pictures of microscopic changes and some better evidences of the histological findings, especially in some of Brissaud's demonstrations of the sensory pathways in the spinal cord.

The author has evidently been captivated by the doctrine of the cerebro-spinal metameres, and possibly has a little magnified its clinical importance. At any rate, he is one of the first in the field to exploit this doctrine, and he applies it to clinical work with a facility that is quite bewildering. For instance, he has devoted many pages to the subject of zona, or herpes, alone, and has sought to illustrate by the distribution of this disease the arrangement of the spinal metameres and their representation on the skin. Brissaud begins his demonstration with a reference to embryology, from which science we learn that the embryo consists at one period of a series of segments, piled one upon the other as it were, and differing little, if at all, from each other. He nowhere admits that this conception of the embryo may be somewhat hypothetical, and that it would practically be difficult to fix upon any exact period of its growth when it could be literally said that all its metameres were identical. On the contrary, Brissaud distinctly defines a metamere as a portion of the embryo, still fragmentary, possessing in itself the whole of the properties and attributes of the being as finally finished or developed. (P. 55.) Our only comment on such a conception of the embryo is that it is distinctly theoretical. In the spinal cord, of course, such a theory is much more conceivable and probable than in the cerebro-spinal axis as a whole, and as Brissaud is dealing here largely with the spinal cord, the criticism is not so important. The metamere is represented all during life by a horizontal section (*tranche horizontale*) of the individual. To distinguish this area from the area of distribution of the spinal roots and nerves (which distribution is usually rather longitudinal, or in the long diameter of the limb or trunk) is apparently the main object of Brissaud's lectures, and for this object he refers voluminously to clinical records, not only of cases of zona, but also of other trophoneuroses, as well as such cord lesions (especially syringomyelia) as sometimes show this metameric distribution of sensory changes. The work in this connection is interesting, and as a rule convincing, but it is embodied in such a diffuse style that it consumes much more labor and time on the part of the reader than should be necessary for a clear understanding of the author's ideas.

On the subject of Brown-Séquard's syndrome Brissaud enters a field where others have been before him—a fact of which he is not suffi-

ciently cognizant. Not a few other observers have noticed this syndrome (or something like it) in cases of trauma and syringomyelia, and records of such cases have been in print for at least four or five years. The most obvious fact in these cases (one which has not escaped Brissaud) is that the dissociation symptom, as commonly seen in syringomyelia, may appear in these cases and be seen on the side opposite the lesion; in other words, the evidence from cord lesions is that the pathways for heat, cold, and pain decussate in the cord at about the level of the entrance of the posterior roots, and that this decussation takes place in the gray matter, the pathways then ascending cephalad by a second order of neurons, whose axis-cylinders probably run in Gowers' tract. The fact that this dissociation may occur as a result of a unilateral trauma was pointed out by the writer of this review in a paper published in this JOURNAL in June, 1894, and again in a report of the microscopic findings of one of his cases in *Brain*, Spring no., 1898. In the latter paper the conclusion was drawn that the fibers for tactile sense ascend in the posterior column without decussating—a conclusion which seems warranted by experiment, as well as by histology, but which is not borne out by some of the reported cases of the Brown-Séquard type. Brissaud accepts the view that the fibers for tactile sense decussate low in the cord, and attempts to explain this hypothesis and to align the theory with cases in which the dissociation occurs. To us his explanation is not satisfactory, although we recognize that the exact course of all the sensory fibers in the cord is still somewhat problematical. There seems to be little doubt, however, that the fibers for heat, cold, and pain pursue the course above indicated, and Brissaud's lectures are devoted to demonstrating that this dissociation can occur in cases of spinal syphilis. If we are to believe with Brissaud that the tactile fibers decussate low in the cord we must accept the view of a double decussation (the second being in the lemniscus); in other words, that some tactile fibers decussate in the cord, and others in the lemniscus. For those who are to follow these obscure subjects, as they are illustrated in the clinic, Brissaud's lectures will be useful; but the reader must not expect to see full reference made to the prior work of others in the same field.

The remainder of the clinical lectures are devoted to miscellaneous topics, of which we need merely mention the reflex of the fascia lata, pseudo-bulbar paralysis, the cephalic trophoneuroses, infantilism, and myxedema.

JAMES HENDRIE LLOYD.

Miscellany.

CRAIG COLONY PRIZE FOR ORIGINAL RESEARCH IN EPILEPSY.

Last year Dr. Frederick Peterson, President of the Board of Managers of the Craig Colony for Epileptics, offered a prize of \$100 for the best original contribution to the Pathology and Treatment of Epilepsy. The seven papers received were submitted to three members of the New York Neurological Society, who gave to the Board of Managers of the Craig Colony the following report:

New York, October 10, 1899.

To the Board of Managers, Craig Colony, Sonyea, N. Y.:

Sirs:—The Committee on the Craig Colony Prize for Original Work in Epilepsy has decided that no award should be made this year. Some of the essays submitted failed to comply with the conditions of the competition; others were more limited in scope than a successful essay should be. Three deserve special mention. Their titles and devices are:

"The Pathology of Epilepsy," by CO $\left\{ \begin{array}{l} \text{NH}_2 \\ \text{OH}, \text{NH}_2 \end{array} \right.$
"Status Epilepticus," by "Aura."

"The Pathology of Epilepsy," by "On and Ever Onward."

Respectfully,

PEARCE BAILEY,

Sec'y N. Y. Neurolog. Society.

GEO. W. JACOBY,

Ex-Pres. N. Y. Neurolog. Society.

IRA VAN GIESON,

Director Pathological Inst. of
the N. Y. State Hospitals.

The prize of 1899 not having been awarded in accordance with the report of the committee, Dr. Peterson now offers a prize of \$200 for the year 1900 under similar conditions. This sum will be awarded to the author of the best contribution to the Pathology and Treatment of Epilepsy. Originality is the main condition.

The prize is open to universal competition, but all manuscripts must be submitted in English. Each essay must be accompanied by a sealed envelope containing the name and address of the author, and bearing upon the outside a motto or device, which is to be inscribed upon the essay.

All papers will be submitted to a similar committee, consisting of three members of the New York Neurological Society, and the award will be made upon its recommendation at the annual meeting of the Board of Managers of the Craig Colony, October 9, 1900.

Manuscripts should be sent to Dr. Frederick Peterson, No. 4 West Fiftieth street, New York city, on or before September 1, 1900. The successful essay becomes the property of the Craig Colony, and will be published in its medical report.

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